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EXAMINATION OF THE CHILD WITH CHRONIC PYELONEPHRITIS

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BOSTON

THE frequency with which a serious hypertension or uremia in childhood is preceded by chronic pyelonephritis renders it essential to use with the maximum effectiveness every medical and surgical procedure of therapeutic value early in the course of the disease, before a progressive hypertension or renal insufficiency has been initiated. In practice many cases will be deprived of early effective therapy unless a thorough examination of the genitourinary system is made with efficiency and dispatch. The organization of the examination is therefore important. For this reason the procedure now in use at the Infants' and Children's hospitals of Boston is presented. It is of course tentative, being subject to change as experience suggests more effective or simpler technics.

CHRONIC PYELONEPHRITIS, HYPERTENSION AND UREMIA

The final clinical and pathologic picture that results from the renal damage in chronic pyelonephritis has been recognized at least since the description of Löhlein[1] in 1917. Longcope and Winkenwerder[2] have recently reviewed the literature and described several cases. In addition, the serious sequelae have been stressed by Abt,[3] Kretschmer,[4] Helmholz,[5] György,[6] Campbell and Lyttle,[7] Ellis[8] and others. Nevertheless, we believe that many cases of the disease are treated inadequately over a period of months or years, possibly because the many cases of acute pyuria that clear up under simple treatment give false hopes concerning the prognosis of the similarly treated chronic case.

We have found no reliable information concerning the mortality rate in chronic pyelonephritis. Gibson[9] in a series of 1,800 autopsies at the Radcliffe Infirmary reports 109 cases of pyelonephritis, or an incidence of 6 per cent. Bugbee and Wollstein[10] in 2,650 autopsies found lesions in the urinary tract in 4.5 per cent. Campbell and Lyttle[7] in 2,420 autopsies found ureteral blockage in 2 per cent and urologic anomalies in 7 per cent.

In 2,043 autopsies at the Infants' and Children's hospitals during the past ten years (1927-1936) we have had 41 cases where chronic pyelonephritis was a primary cause of death, an incidence of approximately 2 per cent. In 26 of these (63 per cent) the patients were under two years of age, while in 15 (37 per cent) they were between two and twelve years. Fourteen of the infants (54 per cent) and 12 of the children (79 per cent) had an anatomic malformation of the urinary tract. Septicemia and infection secondary to the pyelonephritis resulted in the death of 13 of the infants before any significant nitrogen retention developed. Marked nitrogen retention occurred in 13 of the infants and 13 of the children. Two of the children died of hypertensive heart disease without evidence of renal insufficiency. During the same period 296 infants and 489 children with pyuria were treated in the hospitals. Thus the autopsied cases alone indicate a mortality in patients with pyelonephritis under 2 years of age of at least 9 per cent, and in those over 2 years of at least 3 per cent. These figures are raised to 20 and 4 per cent, respectively, if we include the patients who died in the hospitals but were not examined by autopsy. How much these figures would be raised if we subtracted the acute pyurias, confining our statistics to chronic cases, and if deaths outside the hospitals were recorded, is of course unknown.

Of the patients with nephritis admitted to the hospitals during this ten-year period, there were 41 infants and 301 children.** Four of the infants (10 per cent) were examined by autopsy; 3 of these had acute hemorrhagic nephritis, and 1 had congenitally defective development of parenchy-

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**These figures do not include 16 patients with nephrosis.

mal kidney tissue and died shortly after birth. Eight of the children (3 per cent) were examined by autopsy; 5 of these had acute glomerular nephritis, and 3 had chronic glomerular nephritis.

Thus, only 3 patients dying of uremia were proved by autopsy to have chronic glomerular nephritis, while 26 patients dying of uremia were proved by autopsy to have chronic pyelonephritis.

Inadequate as these figures are, they indicate (1) the high incidence of chronic pyelonephritis in childhood; (2) its seriousness in infants with or without an anatomic anomaly; (3) its seriousness in children with an anatomic anomaly; and (4) the fact in both infancy and childhood that it is the commonest cause of chronic renal insufficiency with uremia.

Our experience has, moreover, shown a relation between hypertension and chronic pyelonephritis.[11] During the period 1927-1936 (inclusive), 27 children with both these ailments entered the hospital. Of these, 15 had had them for a period of years without any diminution in renal function as measured by urea clearance or phenolsulfonphthalein excretion.*

With malignant hypertension as a possible sequela of pyelonephritis, the chronicity of the disease becomes even more serious than the usual mortality figures indicate, because many cases classed as nephrosclerosis and heretofore not associated with pyelonephritis may actually follow this disease.

ORGANIZATION OF EXAMINATION

Though ammonium mandelate and para-aminobenzenesulfonamide now provide more effective therapeutic measures[12, 13] than heretofore available, the presence of an anatomic abnormality causing stasis in the urinary tract seriously limits the effectiveness of medical treatment and renders recurrence probable. In many cases where urinary stasis or unilateral infection is present, surgery can relieve the stasis or eliminate the cause of continued infection. To prevent possible delay in adequate therapy a thorough search for an anatomic abnormality should precede treatment. Empirically such a procedure is justified by the high incidence of abnormalities in chronic pyelonephritis,[7] and the long delay in receiving adequate treatment that frequently results from the institution of therapy before a careful examination of the patient has been made. Thus the efficient treatment of this disease usually demands cystoscopic examination, and the information derived therefrom becomes the concern of the pediatrician or internist.

The cystoscopic examination of a small child requires a general anesthetic, and may occasionally be followed by disturbing anuria. It is fraught with difficulties, and the information obtained from it not infrequently falls short of that desired. This information is particularly vital because on it rests the choice between medical treatment and a major operation. Every effort should therefore be made to learn as much as possible from a single examination. The efficient co-ordination of the various examinations and planning of the cystoscopic procedure are essential, if loss of time, undue trauma to the patient and excessive expense are to be avoided.

Intravenous pyelograms furnish data that may not be obtainable by retrograde pyelography. For example they may disclose functional differences between the two kidneys or parts of the same kidney or ureters. They permit the visualization of certain ureteral abnormalities that are obscured by ureteral catheterization. Moreover, they frequently supply the cystoscopist with information which permits him to make a single examination with more success by giving him leads as to what needs further visualization. Or they may permit a more limited cystoscopic exploration, for example, a confining of the catheterization or retrograde pyelography to a single ureter. Thus intravenous and retrograde pyelography are supplementary, not alternative procedures. The visualization of the urethra, bladder and ureteral orifices, and the collection of divided urine specimens from each ureter, are usually so essential that cystoscopic examination is indicated.

Before intravenous pyelograms are taken the patient's kidney function should be known. Since the urine should be concentrated at the time of intravenous pyelography, a concentration test preceding the examination may be used as the measure of renal function. But before restricting fluids in a patient with chronic pyelonephritis, the concentration of nonprotein nitrogen in the blood, or some equivalent for detecting any existing retention, should be determined. Since gas in the intestine may prevent good visualization of the urinary tract, measures should be taken to reduce it to a minimum at the time of pyelography.

During the cystoscopic examination care should be taken lest the observation of the appearance time of any injected dye interfere with the microscopic or bacteriologic examination of the divided urine specimens. Several successive urine specimens from each kidney are required for reliable information concerning the relative function of the two kidneys, as there is great irregularity in the kidney function after the insertion of ureteral catheters. We believe that urine concentration measurements are more significant than excretion rates. If the latter are to be determined, dye that fre-

*For a fuller discussion of these patients and the relation between pyelonephritis and hypertension, the reader is referred to the paper just cited.[11]

quently leaks into the bladder[14] must be included. With bilateral catheterization, any such leakage obviously prevents the determination of the divided function by excretion data. Relative urine concentrations, on the other hand, reflect the respective function of the two kidneys no matter what the leakage.

This information, together with the already determined total renal function, permits an estimation of the function of each kidney. No fluid of any kind is injected through the catheters to the ureters, for if this is done appraisal of the function of each kidney by the concentration of nonprotein nitrogen and phenolsulfonphthalein in small urine specimens becomes impossible. Similarly the injection of the dye should not interfere with the accurate collection of these specimens.

The following plan covering the special examination of the patient with chronic pyelonephritis is being followed at the present time at the Infants' and Children's hospitals.

A. Initial procedures.

1. Examination of vulva and urethra and vaginal orifices for inflammation and discharge.
2. Examination of voided urine.
3. Catheterization for determination of residual urine and for examination and culture of catheterized urine specimen.
4. Observation for incontinence.

B. The determination of kidney function and preparation for intravenous pyelography.

1. The concentration of the nonprotein nitrogen in the blood is determined.*

2a. If this is within normal limits the patient's fluids are restricted as for a urine concentration test, from lunch or supper (depending on the age of the patient) to the time of pyelography the following morning. To minimize the amount of gas in the intestines a very light supper is given.† The patient voids at 10 p. m. and at 6:30 a. m., the volume and specific gravity of the urine from the latter voiding being recorded. If the specific gravity is above 1.020 the urine concentration is enough to assure a sufficient concentration of the dye for its visualization in the functioning calyces, pelves and ureters, and its adequate subsequent excretion. If the specific gravity is below 1.020, another urine specimen is obtained, by catheter if necessary, immediately before pyelography. Should this second specimen have a specific gravity below 1.020, the examination is postponed until after the kidney function has been determined by urea clearance.[15] ‡

2b. If the nonprotein nitrogen is abnormal, a urea clearance test is done to determine the kidney function. If the urea clearance is less than 30 per cent of normal, intravenous pyelography is done only after careful consideration. In patients with markedly diminished renal function the dye is usually not concentrated sufficiently for visualization. Occasionally, however, plates taken several hours after injection of the dye will give a satisfactory opacity when the early plates show nothing. In some cases cystograms may be the procedure of choice.

C. Intravenous pyelography.

1. Immediately before the intravenous injection of the dye a flat plate of the abdomen is taken, developed and examined to see if gas in the intestine is obscuring the urinary tract. If such is the case, an attempt is made to have the gas expelled by the use of an enema or pituitrin and rectal tube. This plate also indicates the size of the kidneys and the presence or absence of calculi or calcified mesenteric glands.
2. The dye (8 to 15 cc. of diodrast for infants and 15 to 20 cc. for children) is injected intravenously, and roentgenograms are taken after 5, 15 and 30 minutes. If the dye is used in patients with impaired renal function, plates should be taken at later periods also. A negative examination up to this point, with the exception of pyuria and bacilluria, and a mild and nonlocalizing history may indicate medical treatment before cystoscopic examination is made. Or the finding of markedly diminished kidney function or inoperable bilateral abnormalities may contraindicate cystoscopy.

D. The cystoscopic examination.

In distinction to the preparation for intravenous pyelograph, the patient should not be on restricted fluids before this operation. Indeed it may be better to keep him on a very liberal fluid intake, so that a relatively large volume of urine is being passed at the time of examination. Such a volume seems to be indicated because the determination of the relative function of the two kidneys is here the functional information desired rather than the maximum function as measured by urine concentrations or excretion rates.

Morphine should be given about 30 minutes before the anesthesia, as this lessens the likelihood of an inhibition of kidney function during the operation.[16]

The following procedure has been adopted as the best for securing the information desired:

1. Thorough examination of the vagina for anatomic abnormality, inflammation or aberrant ureter.
2. Catheterization. The bladder urine specimen is saved for culture, microscopic examination and determination of nonprotein nitrogen concentration.§
3. Examination of urethra for obstruction, inflammation or aberrant ureter.
4. Instillation of saline into the bladder.

*One-tenth of a cubic centimeter of blood is taken by finger or heel prick, and the nonprotein nitrogen is determined by any Nesslerization method adopted for such small quantities of nitrogen.

†Though a light supper and a mild laxative, such as compound licorice powder, with an enema early the following morning if a bowel movement does not result, may frequently result in very little gas in the intestines, no fixed procedure has as yet been adopted.

‡When there are anatomic abnormalities of the urinary tract, urea clearance values frequently vary with abrupt change in urine volume. In such cases the clearance values are more reliable if a large urine volume is maintained before and during the test.

§One to two tenths of a cubic centimeter of urine suffices for the determination of nonprotein nitrogen.

5. Cystoscopic examination of bladder and ureteral orifices.
6. Catheterization of ureters. Care should be taken that all the orifices of the ureteral catheters are patent. The outer ends are plugged to prevent bladder fluid from entering the catheters. After inserting the carefully marked catheters into each ureter, the outer ends of each are placed in sterile tubes for collection of divided urine specimens.
7. A flat plate of the abdomen is taken.
8. After examination of the wet plate has shown the catheters to be satisfactorily placed, the cystoscope is withdrawn, leaving the ureteral catheters in place, and the anesthesia is discontinued. If catheterization can be done without risk of disturbing the ureteral catheters, the bladder is emptied. This prevents reflux of the saline up a dilated ureter and dilution of a ureteral urine specimen. The cystoscopist can now leave the patient with an assistant and a nurse.
9. If several drops of urine have appeared in each test tube, the tubes are removed and set aside for collection of a few more drops later and later for culture and microscopic examination. New tubes, each containing a drop of 10 per cent NaOH, are placed to receive the urine from each catheter.
10. An intravenous injection of phenolsulfonphthalein is made. Its appearance time in each test tube is recorded.
11. If the urine is flowing from the catheters satisfactorily, each test tube is removed when the urine volume in it reaches approximately 1 cc., and is replaced by another for the collection of a second specimen. In case the phenolsulfonphthalein has not appeared, a drop of the alkali is again added to each tube. If possible three specimens are obtained from each ureter. These are saved for the determination of nonprotein nitrogen and phenolsulfonphthalein concentrations. Since the alkali may destroy bacteria, blood cells and casts, the specimens to which alkali has been added are not suitable for culture or microscopic examination.
12. A few more drops of urine from each catheter are added to the respective culture tubes (see Item 9 above). The cultures and microscopic examinations of the urine from these tubes indicate whether the infection is bilateral or unilateral.
13. Finally, the dye is injected into each catheter by the assistant. Pyelograms are then taken. The catheters are withdrawn, the patient is placed in an upright sitting position for 5 minutes, and plates are again taken.

Such an examination should indicate the presence of any anatomic abnormality that might limit the effectiveness of medical therapy. It should indicate whether the abnormality present is susceptible to surgical treatment. It should reveal the presence of a unilateral infection and indicate whether nephrectomy or heminephrectomy is the operation to consider.

One word concerning treatment seems pertinent. If an operation is indicated and performed, the patient should be carefully followed for the persistence or recurrence of infection, and treated promptly if it exists. Indeed, a routine course of sulfanilamide therapy immediately following recovery from the operation and before the patient leaves the hospital is desirable, almost as a matter of routine.

SUMMARY

The mortality from chronic pyelonephritis indicates a serious prognosis for that disease. Of the patients admitted to these hospitals with hypertension or uremia or both many more have had chronic pyelonephritis than have had chronic nephritis. Effective treatment is frequently dependent upon a thorough examination of the urinary tract. It is therefore essential that the examination of these patients be accomplished with thoroughness, efficiency and dispatch. The procedure now in use at the Infants' and Children's hospitals is presented.

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CAUSES OF DEATH IN PATIENTS WITH PEPTIC ULCER

ROBERT T. MONROE, M.D.,* AND EDWARD S. EMERY, JR., M.D.†

BOSTON

THE recent literature on peptic ulcer is virtually unanimous in confirming the theory that it is a chronic disease which goes through alternating periods of healing and activity. Many articles deal with the results of treatment as observed over varying periods of time, but state little about the cause of death. Although accurate figures concerning the fate of patients with the disease can be derived only from a large number of cases followed through life, some approximate idea may be obtained by analyzing the immediately available figures in a similar group.

We have reviewed 1,428 cases of peptic ulcer handled in the Peter Bent Brigham Hospital from 1913 to 1932. We were unable to trace 156 patients; but are fairly confident that few if any of these have died without our knowledge. One hundred and sixty-one of the remainder are dead, or 11.3 per cent of the complete series. But peptic ulcer was the cause of death in only 87, which is 54 per cent of those who died, or 6.1 per cent of the total.

TABLE 1. *Percentage of Deaths Due to All Causes and to Ulcer in 1,428 Cases of Peptic Ulcer in Relation to Sex.*

SEX	NO. OF CASES	ALL CAUSES	ULCER
Men	1,044	12.3	7.2
Women	384	8.3	3.1
Total	1,428	11.3	6.1

Tables 1 and 2 show the incidence of sex and the location of the ulcers. The mortality was more than twice as high in males (7 per cent) as in females (3 per cent). In all cases it varied with the location of the ulcer as follows: gastric, 13.8 per cent; combined gastric and duodenal, 11.5 per cent; duodenal, 4.1 per cent. There were 16 patients in whom the location was not specified, "peptic ulcer" being the only diagnosis made; 5 of

TABLE 2. *Percentage of Deaths Due to All Causes and to Ulcer in 1,428 Cases of Peptic Ulcer in Relation to Location of the Ulcer.*

LOCATION	NO. OF CASES	ALL CAUSES	ULCER
Gastric	210	23.8	13.8
Duodenal	1,147	8.4	4.1
Gastric and duodenal	52	17.3	11.5
Pyloric	3	33.3	0
"Peptic"	16	0	31.2
Total	1,428	11.3	6.1

these died, all from their ulcer. The ulcer was located on the pylorus in 3 patients, 1 of whom has died, but not from the ulcer.

From the Peter Bent Brigham Hospital, Boston.

*Physician to Peter Bent Brigham Hospital.

†Associate in medicine, Peter Bent Brigham Hospital.

The manner in which the ulcer caused death is shown in Table 3. Perforation led, being respon-

TABLE 3. *Apparent Cause of Death in 87 Fatal Cases of Peptic Ulcer.*

CAUSE	NO. OF CASES	PERCENTAGE
Perforation	28	32.2
Hemorrhage	20	23.0
Obstruction	4	4.6
Surgical complications	30	34.4
Undetermined	5	5.7
Total	87	99.9

sible for nearly one third of the deaths in spite of surgical intervention. Hemorrhage accounted for nearly one fourth, and obstruction for less than 5 per cent. In 5 cases, the exact cause could not be determined, although it was obviously the result of the ulcer.

In addition, the table lists 30 patients who died from surgical complications. This item needs further explanation. Although the patients died from various postoperative complications such as bronchopneumonia, the reason for the operation can also be given as the cause of death. For example, 2 patients were operated upon because they continued to bleed under medical treatment; yet bleeding was the real cause of death because it necessitated surgery. Five had obstruction at the pylorus; 2 had both obstruction and hemorrhage. In 5 others carcinoma was suspected. One of these was found to have an ulcer with obstruction, and the case should be classified under obstruction. In 1 patient a cancer developed on a previous ulcer, and 1 case was probably cancer from the beginning. One patient had biliary obstruction in addition to the ulcer, and 1, judging from a radio-

TABLE 4. *Actual Cause of Death in 87 Fatal Cases of Peptic Ulcer.*

CAUSE	NO. OF CASES	PERCENTAGE
Perforation	28	32.2
Hemorrhage	24	27.6
Obstruction	10	11.5
Surgery following medical treatment	9	10.3
Surgery following no medical treatment	4	4.6
Other complications	7	8.0
Undetermined origin	5	5.7
Total	87	99.9

graphic study, had a perforation. Of the 13 remaining members of this group, 9 had surgery because of dissatisfaction with the results of medical treatment, either on their part or that of the physician, or both. Four were advised to have surgery although there were no definite indications for it. Because surgery was elected in these cases the

cause of death must be classified as due to the operation. The actual cause of death in the 87 patients who died of peptic ulcer is given in Table 4.

The age at which patients die from peptic ulcer may give some indication of the severity of the disease. The average age at the time of death for the whole group of 161 patients was 55.9 years. Interestingly enough, it was greater among those who died because of their ulcer (58.3 years) than among those who died of unrelated conditions (52.6 years). The average duration of symptoms for all cases was 12.4 years, and there was little difference in this respect between the patients who died of their ulcer and those who did not. These figures do not give an accurate estimate of the danger of having a peptic ulcer. At present, one can only say that the duration of life in patients dying from it compares favorably with that given for all individuals in insurance tables. Those who die of their ulcer are likely to do so, on the average, about twelve years after the onset of the disease.

At the time this study was begun, it was hoped it would be of service in determining whether patients live longer after medical or after surgical treatment. The figures at our disposal confirm the impression gained from other studies that neither medicine nor surgery cures the disease. Of 52 patients who survived an operation, 31 died of their ulcer at some later date. In other words, the ulcer ultimately killed 60 per cent of the patients who survived the operation, as compared with 51 per cent of 43 patients who received only medical treatment. That is to say, ulcer caused death in a higher percentage of patients who survived an operation than it did in those who were treated medically.

Yet another way to find the comparative value of surgical and medical care as regards prolongation of life and effect on the disease is to take the average age of death following the two forms of therapy. This averaged 63.6 years for the medical cases and 50.2 years for the surgical. While this suggests that surgical treatment was less satisfactory than medical, another possible explanation is that the surgical patients were operated on because the cases were more severe. This is borne out by the fact that there were a few patients in this series whose symptoms had apparently never been severe enough to cause them to seek treatment, and that their average age at death was 61 years.

One is occasionally asked whether it would not be wise to treat all patients with ulcer surgically, and treat medically only those who failed to obtain relief after surgery. The following figures appear to answer this question. Of the 82 patients in our series who were operated upon, in 13 there was no particular indication for surgery. Seven of them died from postoperative complications. These patients would have been better off if they had received medical treatment.

SUMMARY AND CONCLUSIONS

An analysis has been made of 161 patients with peptic ulcer who have died. The ulcer was the cause of death in only 54 per cent. The incidence of death was greater among the males than among the females, and occurred in the following order according to the location of the lesion: (1) gastric ulcer, (2) gastric and duodenal ulcer, (3) duodenal ulcer. Perforation was the most frequent cause of death, followed by hemorrhage and obstruction, in the order named.

An attempt was made to evaluate the results of surgical and medical treatment. Neither method cures the disease. More patients ultimately died of their ulcer after surgery than after medicine. The average age at death was lower after surgery than after medicine, but this was probably due to a difference in severity of the disease. The study suggests that medical treatment should be used for most patients, and that surgery should be reserved for specific indications.

A NOTE ON THE TEACHING OF OBSTETRICS

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THE problem of maternal mortality is of such importance that light on it from any source is welcome. Recent studies in certain cities in the United States have given grounds for the view that the medical profession has a considerable share of responsibility for the disastrous results. Whether it has incurred direct responsibility by not giving good obstetric care, or indirect by not promoting popular education in matters obstetric, is not clearly specified. Whether the mortality in the United States is unduly high has been questioned, but no satisfactory standard of a not-unduly-high maternal mortality has been established.

Accepting as true the general proposition that the maternal mortality would be lower if physicians practiced better obstetrics, the following questions have been raised: Do they practice the best they know how? And, Is the best they know how a reasonable approximation to the best current teaching and practice? These are difficult questions to answer in a satisfactory manner, but it has been thought that a review of the answers given to a simple question in obstetrics asked in a state board examination might prove interesting and perhaps suggestive.

The question appeared in a recent examination set by the Massachusetts Board of Registration in Medicine. It reads as follows:

> Give in detail your treatment of a patient in whom there appeared no sign of separation of the placenta twenty minutes after the birth of the child.

It is hardly necessary to note that this is a perfectly normal case as described, although there is a possibility of complications arising after the twenty minutes have elapsed, and it presents a situation with which any physician practicing obstetrics is likely to be very frequently confronted. Furthermore, it is a not unfair assumption that the treatment is to be carried on until the end of the third stage of labor, that is, until the placenta is outside the body of the mother. It should be said that 250 candidates took the examination, of whom 68 received a passing mark and 182 failed on this question. It should be noted that the treatment was to be given "in detail." No attempt has been made to classify candidates according to graduation from approved or non-approved schools; nor have I tried to present all the extreme vagaries of the candidates or the remarkable combinations of therapeutic procedures.

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A few replies of special interest may, however, be noted. "Patience is the greatest virtue, and not infrequently the relative merits and rewards obtained by being patient are not only fruitful but very satisfactory. With the general condition of the patient being good and no unusual vaginal bleeding being present, there is no imminent or immediate danger to the mother by keeping a watchful eye, so to speak, on the patient and observing carefully and very frequently the state of the uterus and *continue to wait longer.* Hasty attempts to deliver the placenta commonly means many bitter resentful and regretful days, particularly where manual attempts are carelessly made in haste, and inversion of the uterus occurs or even a complete invagination occurs with tragic results, lacerations and profuse hemorrhage." How true! And this is only by way of beginning, yet the candidate actually supplemented his preface by enough correct information to get a passing mark. Another reply is, in part: "Constant twisting of the umbilical cord and reasonable traction may loosen it. If it does not seem to be separating a manual removal is done." Another uses the Credé method only. Another complete treatment is: "The patient be kept strictly in bed, kept warm, the doctor examines patient carefully for the retained placenta. Warm liquid drinks given, also one quarter grain morphine will be considered favorable." One candidate said, "Above everything I would not Credé the uterus," and passed directly from the failure from the administration of pituitrin to manual removal. One advised Credé after waiting twenty-four hours; and another advised Credé, "putting the hand in the vagina and pealing the placenta out."

Thirty candidates noted specifically that there was no occasion for hurry or alarm. Twenty-four said, "Wait," without specifying how long; 20 said, "Wait ten minutes more"; 27 said, "Wait one hour in all"; and varying numbers advised waiting from two to forty-eight hours. In many cases it was not stated what should be done after the period of waiting, but the time to elapse before attempting manual removal is noted below if stated by the candidate. In all, 106 specifically indicated that waiting for varying periods of time was the beginning of the treatment of choice. The others apparently began active treatment at once. Forty-three specified massage of the fundus; 39, traction on the cord; 17, injection of the cord; 183

advised Credé, or some procedure which was not named but closely resembled it. Sixty recommended the use of ergot, hypodermically or by mouth; 110 advised pituitary extract; and many of these candidates used both ergot and pituitrin. Four advised oxytocics without further specification. Ultimate manual removal of the placenta, if necessary, was resorted to by 131, without specification as to time and usually without a reason other than that other methods had failed. In addition, 5 advised manual removal immediately; 1, in twenty minutes; 1, in forty minutes; 34, in one hour; 10, in two or three hours; 6, in six hours; 14, in twelve hours; 4, in twenty-four hours. Forty-three advised hysterectomy for placenta accreta — or "increta," according to 2 candidates. Seven suspected a second fetus. Two recommended hot fomentations; 1, cesarian section; 9, removal of placenta by curettage; and 9, removal by placenta forceps. Six suggested taking the clamp off the cord; 3 limited their treatment to immediate hysterectomy; 1 advised having the patient bear down; 1 advocated the use of a Voorhees bag. There were 9 who agreed that if the placenta did not come away after the Credé maneuver a consultation should be held with another physician.

It is difficult to know just what comment to make on this appalling picture, or what conclusions to draw. The first conclusion might be that many of the candidates did not read the question carefully or did not know the meaning of the words "give in detail." There is also the possibility that the candidates are really as ignorant as they appear to be. It is unsafe to say that the candidates are not given the correct information by their teachers: anyone who is familiar with students realizes in what mutilated fragments information so communicated is returned on examination or quiz. But this question is so simple, the answer is so easy, and the content of the answer should so clearly be a part of the knowledge of every recent graduate in medicine, that at least the following conclusion may be drawn: However excellent the teaching of obstetrics may be, in some respects, on this elementary problem, it has not been effective for this group of candidates.

The teaching of obstetrics in our schools has often been criticized. Perhaps more effective teaching of the fundamentals of practice is one of the lines along which progress will result in the saving of more maternal lives.

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PROGRESS IN LARYNGOLOGY

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THE sympathetic innervation of the nose has been the subject of investigations by Larsell and Fenton,[1] Higbee,[2] Kuntz[3, 4] and St. Ernyei.[5]

Larsell and Fenton[1] take up in detail the pathways of referred pain and the reflex arcs involved. The several possible vasoconstrictor and dilator reflex arcs that are pathways for impulses clearly explain reflex vasomotor disturbances.

The fundamental division of the nervous system is, of course, into the sensorimotor and autonomic systems, and as Higbee[2] emphasizes, the autonomic system is the efferent system, distributed to glands, smooth muscle and blood vessels, and concerned only with vegetative functions.

Kuntz[3] brings out the relation to allergy. He states:

> The autonomic innervation of the mucous membranes and involuntary musculature of the upper respiratory tract, the paranasal sinuses, the middle ear and the auditory tube includes both sympathetic and parasympathetic nerves. The functional state of the mucous membranes, including their secretory and ciliary activity, is regulated and controlled through the autonomic nerves and is intimately related to the functional state of the peripheral blood vessels. Inflammatory reaction differs only in degree from the normal physiological response.
>
> That the autonomic nerves are at fault in allergic disorders is indicated by the sudden appearance of their manifestations. In cases of allergic rhinitis the water content of the nasal secretion is increased and the calcium content diminished. The reduction in the calcium content of the nasal secretion is probably an expression of a shift in the acid-base balance of the body fluids toward alkalinity. The acid-base balance, which is closely associated with the autonomic balance, must therefore be regarded as an important factor in the etiology of allergy.
>
> Therapeutic measures designed to restore the acid-base balance, and consequently the autonomic balance, seem to be indicated in allergic cases and in various other conditions in which the functional activity of the respiratory mucous membranes is disturbed.
>
> Vessels of the cavernous or erectile tissue in the nasal mucosa react to nerve stimulation in a manner differing from that of the vessels in the adjacent mucous membrane.

In regard to pathways of referred pain, Kuntz[4] states:

> Certain pains in the neck, upper thorax and upper extremity which are associated with lesions in the mucous membranes of the nose and paranasal sinuses exhibit the

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characteristic features of referred pains. In view of the anatomic data which indicate that efferent components of the upper thoracic spinal nerves traverse the plexuses on the common and internal carotid arteries and reach the mucous membranes of the nose and paranasal sinuses, these pains may be regarded as referred, in conformity with Head's theory of the localization of referred pains.

Referred pains are accompanied by reflex phenomena which play a part in the causation of the sensations of pain. Impulses directly responsible for sensations of pain probably arise in pain receptors in the peripheral area in which the pain is localized. The stimulation of these receptors is caused by the reflex responses, probably through the accumulation of waste metabolites or the liberation of a stimulating chemical substance.

The mechanisms involved in the components of the referred pains in question which are due to reflex phenomena may be outlined as follows. Impulses arising at the site of a lesion in the nasal or paranasal mucosa are conducted into the spinal cord through afferent components of the upper thoracic nerves, reach the corresponding ganglia of the sympathetic trunks through preganglionic neurons, and are conducted to the periphery through sympathetic neurons. Pain receptors at the periphery are stimulated; the impulses arising in them are conducted into the spinal cord through afferent components of the corresponding spinal nerves and upward through the lateral spinothalamic tract on the contralateral side.

St. Ernyei[5] carried out histologic studies on white rats, guinea pigs and cats. He investigated the finer distribution and connections in the nasal mucous membrane of the olfactory, trigeminal, sympathetic and nervous terminals.

Lyman[6] has summarized the work of Sluder on the sphenopalatine ganglion. Sluder published thirty-five papers devoted to the syndrome of this ganglion.

In the diagnosis and treatment of neuralgia, Hoover and Poppen[7] state:

Trigeminal and glossopharyngeal neuralgias are alike in all respects except the location of the agonizing flashes of pain and the location of the "trigger" areas. The areas in glossopharyngeal neuralgia include the pharyngeal wall, tonsillar area, the base of the tongue and occasionally the ear, while in trigeminal neuralgia they are the mucous membrane about the mouth, lips, nose and various areas on the face. In all cases careful examination should be made of the larynx, upper end of the esophagus, the palate, tonsils and posterior molar teeth, because malignant conditions in these regions may produce symptoms suggestive of a glossopharyngeal neuralgia.

Alcoholic injection is not feasible because of the smallness of the nerve and its very close relation to the vagus, jugular and hypoglossal nerves. Inhalations of from fifteen to thirty drops of trichlorethylene three or four times a day have really been very efficient in giving marked relief from this condition. The surgical treatment of choice is the intracranial section of the ninth nerve in the posterior fossa. This exposure makes it possible to exclude tumors in this area. Following intracranial section of one glossopharyngeal nerve, patients are not conscious of any paresthesia or discomfort whatsoever.

EXTERNAL NOSE

An instance of primary hemangioma of the nasal bone is described by Neivert and Bilchick.[8] This seems to be the first case of this condition reported.

Biegeleisen[9] presents a new treatment for the embarrassing telangiectases of the nose. Until now there has been no satisfactory treatment. The author describes a microinjection technic based upon the treatment of similar lesions on the legs. A special needle is employed to inject 30 per cent sodium chloride directly into the capillary lumen. The author states that the method is safe and cosmetically good.

Benjamins[10] describes several cases of congenital epidermal fistulas and cysts of the nasal bridge. These are illustrated by photographs and by radiograms showing the passages and cysts after injection with lipiodol. The only effective treatment, he says, is complete removal by dissection of the entire fistulous tract and cyst, and this is rendered much less difficult if the lumen is stained by the previous injection of methylene blue.

Weinhold[11] presents 2 cases of infection of the nasal vestibule from vaccine pustules in which the diagnosis was specially difficult and could be made only after a careful consideration of the clinical history. The patients were women; the condition developed rapidly, resembling herpes, acute eczema, or perhaps the more chronic forms of erysipelas. There was some ulceration in the nasal vestibule, crusting, and considerable swelling of the nose and upper lip. The acute inflammation and all the symptoms subsided after about ten days. Both the author's patients were in contact with infants who had been successfully vaccinated some twenty days beforehand.

NASAL AIRWAYS

Buhrmester[12] has continued her studies on the chemical composition of nasal mucin. She finds it to be unlike mucin from other tissues.

Buhrmester and Wenner[13] show that nasal polyps and normal mucous membrane contain a histamine-like substance in approximately equal amounts in the moist tissue. Calculated on the basis of the dry weight of the tissue, polypoid tissue contains more histamine than the normal membrane. Nasal secretion from persons with allergic rhinitis does not have depressor activity when injected intravenously. The hydrolyzed secretion contains much less histamine-like substance than do polyps, calculated on equal protein values. Histaminase is not present in allergic nasal secretions. These writers suggest that the increase in mineral content of the nasal discharge, with its resulting effect on permeability, difference in potential and acid-base equilibrium, is a factor to be considered in studying the formation of polyps and other allergic manifestations.

Chavanne[14] in a long series of experiments on dogs demonstrates energetic limitation of nasal secretion when thyroxin is injected intravenously.

Thyroidectomy seemed to increase the amount of nasal secretion, which could readily be reduced by systematic injection of thyroxin.

Zaritzky[15] describes the changes in the mucous glands and cavernous tissue of the nose due to age.

Certain methods of demonstrating organisms in the nasal mucous membranes, such as microscopic examination of smears from swabbings, tissue sections or cultures, and animal inoculations, have been used by the majority of investigators in the field of nasal bacteriology; however, so far as the literature suggests, the use of epithelial scrapings in demonstrating organisms in the nasal mucosa has been employed infrequently, if at all. Schuster[16] states:

> This method of epithelial scraping offers certain advantages in the study of nasal bacteriology in general, and is of particular value in showing the existence and incidence of tubercle bacilli in the mucosa of the tuberculous.
>
> Of the 50 unselected cases of pulmonary tuberculosis, 30 per cent showed tubercle bacilli in the epithelial scrapings, but in the nasal smear of these same cases no organisms were found. Entrance of tubercle bacilli into the nasal mucous membrane may be an ectogenous invasion caused by fingers, handkerchief or bacilliferous sputum, or endogenous by way of the blood stream and lymphatics.
>
> Patients showing positive tubercle bacilli in the nasal mucosa may possibly be considered as potential cases of nasal tuberculosis.
>
> Since 28 per cent of the positive cases yielded variation or granular forms of the organisms but only 16 per cent yielded rod forms, and since the variation forms are avirulent or dead organisms, and since the latter forms are found where the lytic action or the resistance toward the organism is great, one may possibly conclude that many cases of primary nasal tuberculosis are caused by avirulent or dead tubercle bacilli or their chemical products.
>
> This method of epithelial scraping may be of value in studying the nasal bacteriology in different acute and chronic infections of the mucosa. Since the bacterial flora of the nose is so complex, and since it is difficult to determine which organism is the true infective agent, it is correct to conclude that the organism or organisms found subepithelially are probably the predominating organisms in the disease, and more than likely are the causative factors.

Snyder and Feldman[17] record 3 cases of rhinoliths. All are of the false variety. The first was due to a sponge retained in the nose following an operative procedure. The second showed a shoe button to be the obstructing factor. In the third the nucleus was a piece of twine; in this case the patient, so far as we can ascertain, was the youngest on record with a rhinolith, being only six years old.

Goodman[18] writes on nasopalatine duct cysts. These form in the incisor canal region of the maxilla from epithelial cell rests of a persistent nasopalatine duct. They have no direct relation to the teeth, but in their growth may encroach upon the incisor apices. Such cysts produce an expansion of the bony canal, lined with epithelium and containing fluid. The onset may be insidious, without symptoms, the cysts being discovered on routine x-ray examination of the teeth. Larger nasopalatine duct cysts may give rise to definite pains, which are usually of a neuralgic character and may radiate to the nose or the eyes or along the roof of the mouth. Swelling of the papilla palatina may occur. Tenderness is often elicited on pressure or percussion over the anterior incisor region.

Goodman states that surgical removal and curetting of the areas of the cyst are indicated when repeated swellings of the papilla palatina have occurred or where there is a history of neuralgic pains radiating along the roof of the mouth or the nose or to the orbital region. Removal of the upper incisor teeth in the involved area is rarely if ever necessary since nasopalatine duct cysts are not of dental origin. When, upon expansion, the cyst affects the roots of the adjacent incisor teeth and causes erosion, apicoectomy can be done. In the absence of symptoms or of an increase in size of the area of decalcification caused by the cyst, surgery is not indicated.

The usual site of mixed tumors is the parotid region. These tumors rarely appear in the upper air passages and accessory sinuses. They seem to be most uncommon in the nasal septum. In 1932 Stevenson reported a case of mixed tumor of the nasal septum. He stated that careful search of the literature had failed to reveal mention of such a tumor before his own report. Weidlein[19] has not found any case reported since Stevenson. He gives 1 case in detail. He[20] also lists 3 cases of hemangioma of the septum cured by excision.

Richter[21] advances evidence to the effect that congenital bending of the nasal septum is frequently a result of the primary enlargement of one or several of the nasal conchae. He points out that such hyperplasias occur during the embryonal period, he himself having made observations on human embryos which indicate this. He thinks that such hyperplasia may be of inflammatory or other origin.

Laszlo[22] gives many practical points regarding submucous septum resection. He agrees that the rigid rule that no operation on the septum should be done until the nose is fully developed, usually at the age of eighteen, cannot be strictly adhered to. There are many cases that require an operative procedure as early as eight to nine years of age. In cases where the deviation is very marked and the nasal difficulties are severe, careful removal of the deviated cartilage or bone will be a great advantage to the general development of the child and in turn will help to ameliorate the local symptoms. Removal of parts should be limited to those necessary to establish adequate nasal breathing. Radicalism, which is very important in adults,

should be excluded when one is operating on a developing part of the body.

Ombrédanne and Causse[23] operate even earlier. They contend that the customary diffidence in operating on the deviated septums of children is exaggerated. They supported this contention by bringing before a meeting 2 cases in which they had operated with extremely good results; the ages of the patients were one year and six and a half years. The authors believe that it is bad surgical practice to leave a septal deformity uncorrected when the deformity is obviously interfering with the child's development.

Sternstein[24] studied 32 adults in a series of 140 experiments to determine how much resistance air encounters in its passage through the nasal airways independently of voluntary nasal breathing. A special apparatus and simple technic were employed. The resistance measurements clearly presented a visual record of the amount of obstruction encountered in the nasal chamber.

Moncrieff[25] says that in nasal obstruction of the newborn the urge to breathe through the nose is so strong that not infrequently, when nasal obstruction is present, the baby will die rather than develop mouth breathing in the early days of life. Mouth breathing is not acquired until the age of from ten to fourteen days. Thus in all instances where there is difficulty in breathing in the newborn baby the state of the nasal airways should be carefully examined, and any narrowing be dealt with, if possible, by the passage of a soft rubber catheter.

Van Gilse[26] and Kearney[27] present cases of complete bony atresia of the posterior nares.

Nasal hemorrhage, usually easy to stop, sometimes requires heroic measures. Goodyear[28] gives the etiology and accepted forms of treatment from the simplest to the most radical needed to control hemorrhage of the nose and throat. He[29] reports a case with intractable bleeding from the anterior ethmoid area. Here ligation of the anterior ethmoid artery was effective, and a relatively simple and safe procedure.

Heinberg[30] used an estrogenic substance in postoperative (tonsil and adenoid) hemorrhage in 4 cases, with excellent results.

Goldman,[31] in a series of 42 patients with recurrent epistaxis without any form of blood dyscrasia, treated them with subcutaneous injections of moccasin-snake venom. No other adjuvant treatment was given. The venom therapy was effective in practically all instances either in completely arresting and controlling the nasal bleeding or in markedly diminishing its amount and frequency.

Scal[32] cured a case of severe nasal hemorrhage by radium application alone. Three hundred millicurie hours of radiation were used in each nostril.

COMMON COLD

Dochez[33] details the methods employed to isolate the virus of the common cold, to preserve and cultivate it, and to test its activity in human volunteers. Whalen[34] gives the routine for simple rapid examination of material from the ear, nose, throat, and lower respiratory tract that may harbor pathogenic fungi. He notes the similarity between the pulmonary lesions produced by the tubercle bacillus and by some of the pathogenic fungi, and suggests that the tubercle bacillus may be a yeastlike organism in some phase of its life cycle.

Montgomery[35] emphasizes the relation of climate to diseases of the upper respiratory tract. He feels that the incidence of head colds is definitely related to changes in humidity and temperature. He claims that climate is important in treatment, that the nose is a factor in maintaining the water balance of the body, and that the efficiency of its ciliary and other functions definitely depends upon changes in climate.

Sewall[36] stresses chronic sinusitis as being the endemic focus of the common cold. He maintains:

> The person with chronic sinusitis is the carrier of the common cold. Besides, in "false colds" to which chilling and indiscretions subject him, he is capable of originating within himself, independent of outside contacts, a true, infectious common cold. This is due to the fact that bacteria lurking constantly in his tissues gain potency when his immunity has expired, a true common cold resulting. He is then capable of imparting his cold to others, either by a virus or otherwise, whether they have chronic sinusitis or not. Thus do epidemics of the common cold appear during the colder seasons, which fan into activity the many chronic infections of the sinuses. The person without chronic sinusitis takes "cold" only when the infection is carried to him by one with an acute cold. He never takes cold from chilling. The true cold confers a definite immunity for from six months to a year. The "false cold" is an exacerbation of a chronic sinusitis and confers no immunity.

Use of sulfur dioxide in the treatment of colds is suggested by Rawlins.[37] He found that in what was apparently an epidemic of colds, sulfur dioxide gas inhaled in the first twelve hours of the disease stopped the cold in twenty-four hours in a large percentage of cases.

Instillation of various chemicals into the nose to prevent absorption of viruses and organisms has been tried in the past with poor results. Rake[38] observed that Prussian blue particles pass rapidly from the surface of the olfactory mucosa and within two minutes are found in the tissue spaces, in blood and lymph vessels, in the perineural spaces of the olfactory nerve fibers, and in the subarachnoid space and pia-arachnoid membrane. Preliminary treatment of the olfactory mucosa with tannic acid does not alter the speed of absorption. It does, however, cause an inflammation of the

mucosa, and appears to prevent the pigment from entering the olfactory sensory cells. Both pneumococci and *Salmonella enteritidis* pass through the olfactory mucosa and reach the tissue spaces, the vessels and the subarachnoid space with the same rapidity as the pigment. They invade by passage between the cells of the mucosa, and there is no apparent affinity of the organisms for the olfactory sensory cells. Tannic acid treatment of the olfactory mucosa in no way alters this invasion of organisms through the mucosa. The pantropic virus, equine encephalomyelitis, was detected in the forebrain as promptly as were pigment and bacteria; neurotropic viruses, on the other hand — those of St. Louis encephalitis, rabies and louping ill — were not demonstrated in less than twenty-four hours.

Armstrong and Harrison[39] found that the instillation of various chemicals into the nostrils of mice and monkeys tended to prevent intranasal infection with the virus of the St. Louis type of encephalitis and of the monkeys with the virus of poliomyelitis. Trinitrophenol in concentrations of from 0.32 to 0.64 per cent, either alone or combined with alum, was found to be superior to 4 per cent alum and to be the most satisfactory and efficient experimental agent so far tried by the writers. Trinitrophenol in the concentration and amounts employed was devoid of detectable general or local injurious effects on animals.

NASAL SINUSES

That the development of the nasal sinuses normally occurs by means of a pneumatizing power of the nasal mucous membrane similar to the development of the temporal bone is brought out by Richter.[40]

Richards[41] has devised an apparatus to make repeated measurable observations of the results of transillumination of the maxillary sinus. By means of a photoelectric cell it is possible to measure accurately and make a permanent record of transillumination of the maxillary sinus. This enables one to follow the clinical progress of any given case without repeated x-rays.

In most cases of antral disease the diagnosis can be made with certainty by direct inspection of the nasal cavity, simple x-ray examination, and proof puncture. Sometimes, however, proof puncture gives a negative result, yet the x-ray picture shows a shadow the pathological significance of which may be doubtful. Again, it may happen that the symptoms point to antral disease, while ordinary methods of examination disclose nothing abnormal. In such cases and many others, further information may be obtained by x-ray examination after contrast filling. Kettel[42] introduces the fluid by direct injection. He has examined in this way 100 antrums, the result in each case being verified afterward by operation.

Chronic sinusitis is a common infection in children. Marks[43] considers it one of the major problems in pediatric practice because of its high incidence and its various sequelae. The treatment of chronic sinus disease depends not only on the improvement of drainage from an infected sinus but on the intelligent observance of principles of management applying to a child with any chronic illness. The early recognition of the signs and symptoms of chronic sinus infection and the prompt treatment of this disease syndrome do much to lessen the severity of the illness and the frequency of its complications.

The relation of bronchiectasis to chronic sinus disease is a subject of controversy. Larsell[44] has investigated the lymphatic pathways from the nose. He finds four routes by which material in solution or suspension can reach the bronchial and mediastinal lymph nodes from the region of the paranasal sinuses. These are (1) the trachea, (2) the combined path of the lymph nodes, tracheal lymph duct and blood vessels through the right side of the heart and the pulmonary bed, (3) the purely hematogenous path, and (4) lymph spaces and channels in the visceral cervical space, the dorsal wall of the esophagus, the prevertebral fascia, and related structures, which communicate with the anterior part of the mediastinum. The pathways by which the trypan blue reached the chest from the paranasal sinuses and from the posterior portion of the nasopharynx he describes in detail.

Patients with fever of obscure or "unknown" etiology, especially those with headache, should as a matter of routine have a roentgenographic examination of the paranasal sinuses, intranasal examination, and diagnostic irrigation of the sinuses. Sohval and Som[45] established sinusitis as the cause of obscure fever in 9 cases. The disease in the sinuses was masked in general by the absence of localizing symptoms on one hand and the presence of misleading constitutional manifestations on the other.

As is well known, the occurrence of a positive blood culture in a case of acute mastoiditis indicates the existence of a complication. This is also true in acute sinus disease, according to Goldman.[46] In a series of 300 cases he finds that, except for an occasional case, the occurrence of a positive blood culture in acute sinusitis points to the coexistence of a complication or some associated systemic infection. His explanation of this failure of acute paranasal sinusitis to develop bacteremia unless followed by complications is as follows:

> The veins that drain the mucous membrane of the paranasal sinuses and penetrate their bony cells may be so

small that they become thrombosed and sealed off before organisms can enter the general circulation. When a focus is set up in the skull, meninges or orbit by contiguous extension from a sinusitis, however, invasion of the blood stream may then occur from an infection of the larger veins which drain these sites. These veins, because of their larger size, do not thrombose so easily, and are able to feed the general circulation repeatedly with showers of bacterial emboli.

Kramer and Som[47] report 10 cases of sphenoid sinusitis with meningitic symptoms, 6 of which terminated fatally. The authors discuss the symptoms, diagnosis and treatment of these cases. They believe that the intracranial involvement is predominant, and that the sphenoid sinus is likely to be overlooked as the basic lesion, and add that these cases are unlike those of uncomplicated sphenoid sinusitis.

Wattles[48] reports a case of benign giant-cell tumor of the ethmoid labyrinth. This is a rare disease, only 7 cases having been previously reported.

Arons[49] emphasizes the benign character of an ossifying fibroma of the maxillary sinus. With an original diagnosis of osteosarcoma, radium therapy was followed by x-ray. There has been no recurrence after ten years.

Mithoefer[50] discusses the nonoperative treatment of sinus disease.

Fitzhugh[51] finds that neosynephrin hydrochloride produces constriction of longer duration than that caused by ephedrine hydrochloride, and that there are no apparent undesired effects.

The frequency of infection with the staphylococcus in sinusitis and early bronchiectasis is noted by Woodward.[52] He advocates treatment along specific lines with staphylococcus toxoid and antitoxin, in addition to the older, well-recognized methods.

Bryant[53] believes that fever therapy augments the vascular phase of inflammation and that it is of value in the treatment of acute sinusitis.

Van Alyea[54] has made a study in 164 cadavers of the surgical accessibility of the osteum of the maxillary sinus. In half of his specimens canalization of the osteum was impossible or very difficult, so that from a mechanical standpoint puncture is the more efficient method. He states that a cavity may be more rapidly and thoroughly flushed out when separate openings are used for intake and outflow.

Shea[55] and Miller[56] detail their operative procedure in establishing permanent openings into the antrum through the nasoantral wall.

A review of the published investigations on regeneration of sinus mucous membrane after operative removal has enabled Brownell[57] to say:

> From the microscopic and macroscopic observations described, one is drawn to the inevitable conclusion that complete regeneration of the lining of the paranasal sinuses, including ciliated columnar epithelium, is the rule after the operative removal of the original membrane. The time required for complete regeneration of the epithelium is apparently between three and five months. The type of cells composing the regenerated lining depends, in part at least, on its source, for while the epithelium is usually of stratified ciliated columnar type and comes from the nasal mucosa, it may be of stratified squamous type and come from the buccal mucosa.

Articles on the technic of radical surgery of the frontal and ethmoid have been written by Howarth,[58] Yates,[59] Sanson,[60] Reeves[61] and Hajek.[62]

OSTEOMYELITIS OF THE FRONTAL BONE

Because of its insidious onset, the paucity of external symptoms and its course, leading to brain abscess and death, osteomyelitis of the frontal bone is one of the most dreaded complications of infection of the frontal sinus. In the past few years, following the lead of Furstenberk and Mosher, real progress has been made in combating and overcoming this disease. Its pathology, bacteriology, symptoms, signs and treatment are becoming better understood. During the past year papers have been published by Mosher,[63] Fincher,[64] Jones,[65] Williams and Heilman,[66] Behrens,[67] Adson and Hempstead[68] and Lemon.[69]

The predominating organism has been found to be *Staphylococcus aureus*. But it may be found with increased use of anaerobic cultures that this is not the true etiologic agent. Williams and Heilman[66] say:

> The finding of the identical organism, an anaerobic streptococcus, in two cases of osteomyelitis of the frontal bone, together with the apparently unusually favorable results obtained by specific therapy with an autogenous antivirus, is suggestive that the organism causative of this lesion may have been isolated. This hope is intensified by the fact that previously there has not been any adequate explanation as to why in an occasional case of frontal sinusitis this lesion should develop either spontaneously or after operation, when the bone is equally exposed to infection after any type of infection of a sinus or intervention which involves it. The conjecture that the disease is of staphylococcic origin has not seemed satisfactory, because staphylococci are frequently present in sinuses which have been operated on without producing this type of pathologic change. We present our findings with the hope that they will be confirmed in further investigations by ourselves and by others. Treatment with the antivirus clinically seemed to have a favorable effect in these two cases.

The mortality rate, however, is high. Lemon,[69] in reviewing 91 cases of osteomyelitis of the frontal bone, found the mortality to be 51.6 per cent; of these deaths, 55 per cent followed an acute infection and 45 per cent a chronic infection. Brain abscess was a complication in 30 per cent of the 91 cases.

It may be, as Behrens[67] suggests, that there are two types of cases, the localized or self-limited type, and the progressive type, but the latter is the one usually seen in this part of the country. It is this type that demands radical surgery, which must be prompt and thorough. All surgeons, of course, stress the value of supportive measures. Blood transfusions, bacteriophage and vaccines are to be used throughout the course of the disease. But conservative measures alone are inadequate. Radical operation — multiple radical operations if necessary — offers the best chance of success.

Mosher has outlined a clear, orderly, logical method of procedure in these cases. His work is firmly supported by pathological study. In his latest paper[63] he concludes:

> In reviewing all the cases which we have had at the Infirmary, especially those of the past five years, our operative results have been progressively better, and better in direct proportion as the operations performed were systematic and radical.
>
> I feel even more strongly than I did in my original paper of three years ago that the edema of the skin of the forehead is a rough guide to the extent of the bone and periosteal infection. Further, as was pointed out in that paper, if there is actual bone necrosis the bone is infected without necrosis for an inch to an inch and a half beyond the necrotic area. Bone necrosis does not occur until seven to ten days after the pitting edema appears, and the x-ray is not positive until necrosis appears. Examination of the bone specimen removed in two of the cases just reported showed that the infection spreads along the inner surface of the bone, as well as by the diploic veins. When the infection spreads by way of a diploic vein it may localize at a point far from the original source of infection. When it does so localize, the pus tends to work both inward and outward, giving either a subperiosteal abscess or an extradural abscess, or both, with a destruction of the bone between the two. When a case has lasted two or three weeks, the operator should expect to find one or both of these conditions.
>
> The more I see of osteomyelitis of the frontal bone, the more I feel that the whole face of the frontal bone should be removed as a routine from the hairline to the eyebrow. Preferably, it should be removed in one piece. However, if the patient is in poor condition and there is an area of necrosis, it is justifiable to work from the necrotic area outward, removing the bone for an inch to an inch and a half in all directions from the necrotic area. I believe further that both frontal sinuses should be opened, and the anterior and posterior walls of each sinus removed. I feel strongly that the lateral limit of the bone flap on each side should be at least the outer angle of each frontal sinus, or better, the outer angular process of the frontal bone on each side. The objection to this extensive removal is the deformity. It has been proved that fully 90 per cent of this can be corrected by modern plastic surgery. Therefore the surgeon should not allow his hand to be halted by the question of deformity. If he does, he will lose most of his cases of osteomyelitis of the skull.

HAY FEVER

Coke[70] describes methods by which cases of paroxysmal sneezing can be separated into allergic groups: microbic groups and those which are primarily of vasomotor origin. Suitable treatment is suggested for each group.

Clarek and Rogers[71] regard allergic rhinitis as the initial and simplest form of a general allergic disease of the respiratory tract, occurring equally in the two sexes. Allergic rhinitis responds well to allergic treatment. For some reason these authors find that men are more likely to have the more serious forms of the disease — asthma and hay fever.

According to Linton,[72] intramucosal tests are superior to intradermal tests in allergic rhinitis. He finds that the reactions to intradermal tests are not a reliable index of the sensitivity of the nasal mucous membrane. The offending allergens, however, in most persons with negative cutaneous reactions can be determined by intramucosal tests.

There is lack of agreement as to the nature of the active substances of pollens; some experimenters believe them to be albumins, while others think that they are of small molecular structure. Benjamins[73] concludes from his experimental work that the active substance of pollen is not a protein but a small molecular substance.

The importance of molds in seasonal hay fever and asthma is brought out by Feinberg.[74] His conclusions are as follows:

> 1. The ever present spores of fungi in the air are common causes of respiratory allergy.
> 2. A great tendency exists for many of the mold-sensitive patients to assume a seasonal character in their symptomatology because of the greater prevalence of mold spores in the warm months.
> 3. The evidence for the etiologic factor of molds in the group discussed consists of positive skin and passive transfer tests, the history of a "season" which is not in entire conformity with the pollen reactions, the clinical observation of the patients and comparison of their symptoms with reference to daily pollen and mold counts, the failures in pollen treatment, and the good results and a few systemic reactions with mold desensitization.
> 4. This group consists of such a definite unit that with some experience most of these patients can be catalogued before sensitization tests are made.
> 5. Of twenty-eight patients treated by specific mold desensitization and observed during at least one season, twenty-three obtained satisfactory results.
> 6. The conclusion cannot be escaped that seasonal hay fever and asthma due to molds is a definite and important allergic entity, second in importance only to pollen disease.

Zinc ionization (iontophoresis) has not yet established its true place as a form of treatment. Harkness[75] explains why he does not as yet use iontophoresis. This, he says, is a local symptomatologic treatment, perhaps a form of cauterization. The symptomatology is definite and the procedure simple. Facilities for interpreting results are easy. Individual technic should not be a material factor. But the author cites reports from the current medical literature, and offers a strong protest be-

cause according to it the relief obtained varies from zero to 100 per cent. He adds that as regards the harm that may be done, the various methods of carrying out the treatment, and so forth, there is similar disagreement.

Dean[76] states definitely:

> Ionization does improve most cases of allergic rhinitis. The less the sensitivity to allergens, the greater the relief. Ionization does produce deleterious results in the mucous membrane of the nose which last at least two years. Many changes are permanent.
>
> There is no excuse for using ionization if procedures that do not produce deleterious results will give a satisfactory result. Ionization should not be used unless the benefits derived are greater than the deleterious results produced.

Jarvis[77] stresses the use of suprarenal concentrate in otolaryngology. He finds it of value in treating edema of unknown etiology, urticaria, angioneurotic edema and hay fever.

Scott-Brown[78] discusses the technic and use of radium in recurrent polyps of the allergic type. He achieved "dramatic" results in 14 cases.

OZENA

The etiology and treatment of this disease remain unsolved problems. Kurata[79] carried out experiments in rats that enabled him to conclude that it is a deficiency disease due to lack of vitamins and sunlight. The specific symptoms appear at puberty, owing to rapid development of the "sympathicoendocrine" glands. Reichardt[80] reviews the literature on the problem of inheritance of ozena, and reports studies in 27 families in which the disease occurred. He thinks that the condition is inherited as a dominant mendelian characteristic.

Amersbach[81] gives his views on the pathology and treatment of ozena, based on twenty years' experience. During this time he treated some 400 cases, and most of them were followed up for long periods. The genuine forms of atrophic rhinitis, he states, must be clearly distinguished from the secondary forms, for example, those in tertiary syphilis, and after severe trauma of the nose. The theory that ozena is caused by chronic sinus suppuration has lost support. Such forms of atrophic rhinitis are often associated with a sclerosis of the face bones and a poor development of the accessory sinuses. This change in the bones is not secondary to ozena, but is due to an interference with free pneumatization, a function of the mucous membrane. There is a constitutional inferiority (*Minderwertigkeit*) of the mucous membrane which is the cause of both atrophic rhinitis and of the poorly developed sinuses. Amersbach explains the pathology in the same way as that of the sclerosed mastoid in chronic middle-ear suppuration. The term "sclerosing ozena" is, he holds, a misnomer.

He states that the various theories which attribute ozena to endocrine influences, or which have linked the etiology with certain chemical states of the blood, such as cholesterin deficiency, alteration of the calcium level, and so forth, have not withstood subsequent critical analysis. Specific infection theories (Perez, Abel-Lowenberg) are also being gradually abandoned, and with them vaccine therapy.

Lautenschlager's operation for ozena consists in mobilizing the lateral nasal wall and pushing it toward the septum. The good results, he believes, are entirely due to the extensive formation of granulations and the temporary increase in the circulation of the part, associated with the operative trauma. The more the outer wall is broken up during the operation, the better the result. The relief from symptoms is only temporary, and they have usually returned when the patients are seen again in after years. The same arguments, in Amersbach's opinion, apply to the various implantation proceedings. A wide nasal cavity per se is not a causative factor in atrophic rhinitis. Many persons have wide noses with a normal mucosa.

Zakrzewski and Wiza[82] found bacteriophage therapy good in controlling the disease, but noted relapses in all of 13 cases a few weeks after cessation of treatment. Colloidal manganese, administered intramuscularly and by mouth, was used by Mörch.[83] He examined 25 patients at periods of from one to seven and a half years after their courses of injection, and found permanent disappearance in 14 cases. There had been temporary success in 8 others.

Stovin[84] has found galvanic current an excellent form of treatment. He says:

> The stimulation of the mucous membrane produced by the galvanic current tends to restore its normal physiologic function. In most cases of ozena, the loss of the foul odor after this procedure is striking. In simple atrophic rhinitis a notable result is the restoration of the sense of smell. This treatment of course does not narrow the nasal cavities, but it restores the membranes to a healthier appearance and improves the sense of smell.

MOUTH

Lesions of the tongue, lips, gums and palate occur frequently, and are the forerunners of a serious condition known as alcoholic pellagra, which is probably a deficiency disease. The term stomatitis in general covers the condition. The mucous membranes become deeply reddened, swollen and tender, and here and there ulcers of a mild type develop. Vincent's organisms are easily demonstrated. In a series of over 200 patients suffering from chronic alcoholic pellagra, 60 per cent were found by Blankenhorn and Spies,[85] to have these lesions of the mouth and tongue. According to these authors early treatments con-

sisting of a nutritious diet, wheat germ, and extracts of yeast and liver result in marked improvement in from twenty-four to thirty-six hours, and within a few days the tongue and mucous membranes have returned to their normal condition.

The five steps in treatment of carcinoma of the lip stressed by Hollander[86] are these: (1) establishment of the diagnosis through examination of the local lesion and serologic tests of the patient to exclude syphilis; (2) correction of oral sepsis; (3) treatment of the local lesion with unfiltered roentgen rays, in massive doses to produce blistering; (4) treatment of the cervical lymph nodes, whether palpable or not, with heavily filtered deep radiation; (5) aftercare, including careful follow-up to make sure that the patient does not return to the use of tobacco and to habits of poor oral hygiene, examination of the lymph nodes and periodic blood counts.

It should be emphasized that 98 per cent of all cancers within the buccal cavity or on the lips are said to occur in individuals who neglect their mouths. In the last five years cancer of the mouth in white women has increased 45 per cent in Georgia, possibly because of the increase in cigarette-smoking. Campbell[87] concludes: "Cancer of the mouth is hard to cure but is easily prevented by proper care of the teeth, avoidance of excess use of tobacco, and prompt treatment of leukoplakia and chronic irritations of the tongue or lips."

Ward and Betts[88] use radium capsules in epitheliomas of the lip, and follow this with fairly heavy x-ray treatment, only applied to the neck if there is definite evidence of metastasis.

The relief of pain during irradiation treatment of oral cancers can be secured by alcohol injections of the second and third divisions of the fifth nerve. This allows the patient to wear the necessary protective shield more comfortably, makes cleansing of the mouth easier, and permits better nutrition during the period of increased radon reaction. The technic of injection is described in detail by Hare, Poppen and Hoover.[89] The second division of the fifth nerve is injected for lesions which involve the hard palate and anterior three fourths of the soft palate, the maxilla and the upper portion of the cheek. They inject the third division for lesions of the tongue, the floor of the mouth, mandible and lower portion of the cheek.

True aberrant goiter in any location is seldom observed. Rosedale[90] reports a case of the "tongue root" type of intralingual thyroid presenting far anteriorly in the tongue, together with the results of its assay for iodine and a detailed description of its gross and microscopic pathology.

The incidence, pathology, signs and symptoms, complications and treatment of intralingual thyroids will now be discussed in relation to some of the pertinent literature.

Buckman's[91] conclusions are as follows:

1. Lingual thyroid tumor is a developmental abnormality presenting at the base of the tongue, or within or beneath the tongue, resulting from a vestigial rest of the thyroid anlage.
2. The precaution should be made of ascertaining the presence or absence of other thyroid tissue in the normal location before removing the lingual growth for the relief of symptoms.
3. The lingual tumor may produce grave dyspnea, dysphagia or hemorrhage, but is unlikely to undergo malignant change.
4. It usually appears in the female, at the several periods of endocrinal hyperactivity: puberty, pregnancy and the menopause.
5. Removal of the lingual thyroid may be, and often has been, followed by myxedema. The postoperative appearance of tetany has been reported, and points to the association in development between the thyroid gland and the parathyroid.

Laryngeal thyroid is extremely rare, as rests of thyroid tissue there are outside of the usual course of embryonal descent. Beeson[92] reports a case of aberrant intralaryngeal thyroid tumor, with recovery and nonrecurrence, in a man fifty-one years old.

Lipoma of the tongue is very rare. Mazzini states that only 1 case occurred among 259,366 patients seen during thirty-one years. There are 44 authentic cases in the literature, to which a case reported by Smith[93] is to be added. A forty-five-year-old woman had a lipoma of seventeen years' duration removed by him. The histologic report was "simple lipoma." The mass weighed 32 gm. and measured 11 by 9 by 7 cm. This is the largest of these tumors to be reported.

Spencer[94] reviews the literature and reports 14 cases of advanced carcinoma of the tongue. The patient who survived operation the longest lived three years, four months and nineteen days. He feels from a review of the literature that definite progress is being made in the treatment. The disease is recognized earlier. Broders's classification is a real help in outlining treatment. Surgical diathermy has been a definite improvement over cautery, although it has not displaced cautery entirely, and should not. Irradiation by high-voltage roentgen rays is an improvement over low-voltage irradiation, just as the radium bomb or teleoradium is an improvement over the use of small quantities of radium. The recognition of the importance of syphilis of the tongue in causing cancer of carious teeth with poor dental hygiene he believes will do much to prevent and relieve the suffering from lingual carcinoma.

SALIVARY GLANDS

Congenital absence of salivary glands is evidently a rare abnormality. Cases of absence of the submaxillary glands alone have been reported by Gru-

ber in 1885 and Bruno in 1894. Nayak in 1926 published a case of congenital absence of the parotid glands, and Ramsey has reported absence of salivary secretion in a father and daughter both suffering from dental caries.

A case of congenital dysfunction of the salivary glands is reported by Sharp.[95] These were present but atrophic. Dental caries and atrophy of the mucous membrane of the mouth and tongue were observed, as in all previously reported cases. No abnormality of the lachrymal apparatus was detected apart from the fact that the boy was never seen to shed tears. There was no hereditary factor in the case. The boy was eight years old, and the remainder of his examination was essentially negative.

A case of Mikulicz's disease is reported in very great detail by Aboulker.[96] His conclusion, which is backed by a biopsy of the parotid gland, is that Mikulicz's disease is a cirrhosis of the lachrymosalivary system. It is a dystrophy of unknown cause not unlike certain hepatic cirrhoses the origin of which cannot be accurately determined. Prognosis in these cases is not bad, but treatment must be prompt and thorough. The most satisfactory form of treatment is radiotherapy. By this means the lymphoconjunctival neoformation is absorbed, and this allows that part of the gland which is still alive its liberty of function.

The facts that parotitis is relatively common and that calculi in the parotid are relatively rare, whereas the opposite obtains in respect to the submaxillary gland, are well known. The reason for this is probably the difference in the secretions of these glands. That of the parotid is poor in mucin, whereas the submaxillary saliva is rich in mucin. The fact that mucin readily dissolves mineral salts and is strongly bactericidal probably accounts for this.

Ruppe, Lattès and Voisin[97] present 5 cases of parotid lithiasis. They state that the disease may occur at any age, manifesting itself by swelling and pain, and sometimes leads to suppuration. Abscesses which occur in connection with Stenson's duct anterior to the masseter are the most favorable, as these often discharge into the mouth. Those occurring more posteriorly in the gland may lead to a salivary fistula and are best treated by radiotherapy. They believe that diagnosis with lipiodol helps to differentiate intermittent parotitis, cysts of Stenson's duct and parotid abscess.

The value of lipiodol should be emphasized; it has enabled us to make great progress in our diagnosis of salivary gland disorders. For this reason, Csillag's conclusions[98] in regard to the use of a radio-opaque medium are here quoted in full:

> In addition to calculi, which have hitherto been regarded as the most frequent cause of intermittent swelling of the salivary glands, account must be taken of obstruction due to pathological changes in the ducts themselves. Acute inflammation of the glands, as in mumps, is sometimes followed by connective tissue thickening (fibrosis), with strictures of the ducts. Inflammatory diseases of the mouth may lead to catarrh of the salivary ducts, with swelling of their mucous membrane, and thick secretion which blocks the already narrow lumen. There may thus be formed localized dilatations of the ducts (sialodochitis cystica), which tend to become more numerous and larger the more often the inflammatory attacks (parotitis intermittens acuta) are repeated.
>
> The diagnosis of these conditions has been rendered possible by x-ray examination with "contrast filling" (sialoadenography), and there remain few cases of swelling of the salivary glands the cause of which cannot be determined by this means, together with clinical examination (for example, the use of a sound).

This author reports 8 cases (6 women and 2 men) of intermittent salivary gland swelling, in 4 of which obstruction by calculus could be excluded by contrast filling with lipiodol and sialoadenography which showed that the salivary passages were free from obstruction, and the diagnosis of regional lymphadenitis, probably of tonsillar origin, was thus confirmed. In the 4 other cases the sialoadenogram showed widening of the ducts and ectasia of the smallest canals, so that the x-ray picture suggested a cluster of grapes. There was reason to suppose that in each of these cases the condition was the result of patches of cicatricial thickening in the duct system, originally due to epidemic parotitis. "Contrast-filling with lipiodol," he says, "was of practical value not only in the diagnosis, but also in the treatment of those patients in whom there was disease of the ducts or glands."

Adenoma of the parotid is relatively rare — so rare, indeed, that its occurrence has been doubted. However, since the question of its validity was raised twenty-odd years ago some 15 cases have been recorded, to which Kunstmann[99] adds 4 of his own. He recognizes an acinous and a canalicular type. The former arises in the glandular portion of the parotid, is lobular in outline and encapsulated, and, like the parotid of the newborn, secretes mucus, though this feature has received but little attention in previous descriptions. Adenomas of the canalicular group, to which 3 of his cases belonged, are neither lobular nor encapsulated, and are distinguished by a profuse secretion of mucus with extensive cyst formation and papillary outgrowth. They originate in solid undifferentiated buds from the ducts, and sometimes consist rather of large alveoli. Carcinomatous change has not yet been encountered. He adds that the few solid adenomas reported in the literature probably belong in the acinous class, though the possibility that there exists a pure, solid, canalicular adenoma cannot be dismissed.

Harris[100] describes a tumor at the angle of the right jaw in a man of seventy, which was removed with the parotid gland. He believes this to have been a parotid gland adenoma arising from the duct cells. It closely resembled the tumor reported by Gruenfeld and Jorstad[154] and described by them as an onkocyte tumor.

The occurrence of pulmonary metastases from tumors of the mixed salivary group is not rare, as evidenced by the report of 3 consecutive cases by Olson.[101] He feels that the clinical and pathological study of these tumors is of little prognostic value because the degree of malignancy cannot be accurately determined, and advocates routine roentgen studies of the chest before therapeutic measures are undertaken.

TONSIL

Minear, Arey and Milton[102] have made an interesting study of the development and form of the crypts of the palatine tonsil. Their findings are as follows:

> The crypts of the human palatine tonsil begin to appear during the third fetal month as solid ingrowths from the epithelial wall of the tonsillar fossa. Subsequently these epithelial processes grow, branch and canalize, although the end of such progressive development is not reached until late childhood.
>
> Although the maximum number of crypts is reached during childhood, these later elongate and enlarge by interstitial growth to form the definite crypt system.
>
> The approximate area of the epithelial lining of the adult crypt system of one tonsil was calculated to be 46 square inches (295 square centimeters), whereas the exposed surface area of an entire pharynx was only 7 square inches (45 square centimeters).
>
> Any tendency to empty the tonsillar crypts through natural or artificial means must necessarily be highly inefficient, owing to anatomic constrictions and the tendency of the contents of a complex convergent system to impact at the bottle-neck region of the main crypt. Such plugging is further enhanced by the circumstance that the main drainage channel is often smaller than its tributaries.

Histopathologic studies of the tonsil by Meeker[103] bring him to the conclusion that mucous glands open into crypts and are closely related to tonsillar lesions, such as peritonsillar abscesses. He thinks that the true lymphoid tissue found beneath the main tonsillar mass in the adult is an extension along mucous glands (not along blood vessels). The recurrent tonsil in the adult arises from residual tissue about the mucous glands. He finds a constantly recurring group of mucous glands associated with a bar of cartilage. These are always in the same location, in the constriction between the middle and lower lobes of the tonsil.

Urbantschitsch[104] histologically examined the tonsils and adenoids of 446 patients. All specimens examined showed alterations due to chronic inflammation. In 7 cases deposits of lime salts were seen, and in 33 extensive fungus colonies. Epithelioid cell tubercles were present in 5 cases (the adenoids were affected in 2) where there was no evidence of a general infection or any suspicion of tuberculosis macroscopically. In 2 cases (a man, aged thirty-five, and a girl, aged ten), a carcinoma was proved to be present in the tonsils. Macroscopically the tonsils were of similar size and showed no external evidence of neoplasm. In both patients the chemical blood test (Freund-Kaminer) was positive.

Seventy-five pairs of tonsils removed by tonsillectomy were examined by Menzel[105] both histologically and bacteriologically. Professor Lowenstein obtained tubercle bacilli in pure culture from the tonsillar tissue in 5 cases. Histologically, definite tubercle formation was found in only 1 case, which incidentally belonged to the series from which positive cultures were obtained. He states that in all probability there is a generalized tuberculous infection of the body which results on the one hand in the presence of bacilli in the tonsils, and on the other in a joint rheumatism exhibiting an allergic phase of the infection, in-so-far as in all cases positive lung findings were obtained.

Smith[106] presents a correlation of the history leading to the tonsillectomy, the pathological diagnosis of the tonsils and the subsequent clinical course over a period of eight months. From this study it appears that in a routine group of patients the percentage of persons who give evidence by history or clinical examination of infected tonsils should be between 85 and 90. In this series the microscopic examination of the slides revealed that 86 per cent had tonsils with a pathologic change. He continues that tonsillectomy and adenoidectomy have proved beneficial in cases in which a focus of infection was suspected in the tonsils and in which there was a history of recurrent colds and sore throats, aural involvement, nasal obstruction, cervical adenitis and enlarged tonsils. The procedure seems to be of questionable value in cases of indigestion, laryngitis and pain in the throat. The so-called immediate results of tonsillectomy, as noted in this study, are compared with the less favorable results after a period of ten years, as shown by the figures of Kaiser.

Acute rheumatism is usually caused by a streptococcal infection, according to Barwell,[107] and the source of the infection is in the lymphoid tissue of the throat. Its incidence is certainly diminished by tonsillectomy. Owing to the numerous scattered nodules of lymphoid tissue in the pharynx, however, tonsillectomy is not certain in its prevention, although it makes recurrence less probable.

Of 1112 patients with hay fever and asthma,

Waldbott and his associates[108] performed tonsillectomy in 228 before the onset of allergic symptoms and in 205 after they had developed. In the latter group, tonsillectomy resulted in definite relief in 1.9 per cent, temporary relief in 1.9 per cent and aggravation of the allergic symptoms in 11.6 per cent. In a control group of 60 patients in whom tonsillectomy was performed after they had been under the author's care, the results were substantially the same. Tonsillectomy performed for conditions other than allergy was successful in 35.2 per cent of the allergic group, in 36.4 per cent of the "preallergic" group, as compared with 72 per cent improvement recorded in normal controls (Kaiser). In the 228 patients in whom tonsillectomy was performed before the onset of allergic symptoms, 14.4 per cent developed allergic manifestations within three months, 29.3 per cent within six months, and 48.6 per cent within two years after operation. This compares with 26 per cent showing frankly allergic symptoms within three months, 47 per cent within six months and 63 per cent within two years in patients whose tonsils were removed for the relief of "nasal colds" and "bronchitis." The greatest benefit from tonsillectomy was obtained in the earlier age groups. The operation was practically always a failure in patients more than twenty-five years old. The efficacy of the technic of removal did not affect the result. The operation was less successful when performed during the pollen season. Although the results of tonsillectomy were disappointing, these authors believe that it should be performed in patients having frequent infections. In others with allergic catarrh of the upper part of the respiratory tract which may or may not involve the tonsils, they believe that tonsillar tissue is a definite asset to the system and should be preserved.

Jackson, Parker and Brues[109] found 28, or 11.8 per cent, of 236 cases of malignant disease of the tonsil to be malignant lymphomas of one type or another. Nine additional cases from other sources are included in their study. The greatest incidence was in the seventh decade; and 62 per cent of the cases occurred between the ages of forty and sixty-nine. Five patients were in the second decade. The sex incidence was 57 per cent in males, 43 per cent in females. Sore throat was the commonest initial symptom, occurring in 14 cases; enlargement of the cervical nodes (11 cases) and swelling in the throat (6 cases) were next in frequency. Subsequent symptoms developed rapidly, causing patients to seek medical aid in an average of four months from the onset of the first symptom. The tumor invariably had markedly enlarged the tonsil. In 67 per cent, either localized or generalized metastasis had occurred when the patient was first examined. The prognosis is poor, only 8 patients of this series being alive. Of these, 1 is alive after eighteen years, 2 after ten years and 1 after five years. The duration of life averaged two and a half years; it varies directly with the presence of localized or generalized metastases; namely, the more general the involvement, the worse the outlook. In 44 per cent of the patients who died, the disease was localized either to the tonsils or to the cervical nodes; in 56 per cent it had become generalized. The response to radiation is no index as to prognosis. They suggest that in the future the treatment should consist of very heavy initial radiation with comparable doses for recurrences.

Peritonsillar abscess has received considerable attention during the past year. Neffson and Brem[110] call attention to the fact that clinical diphtheria may sometimes be easily mistaken for it. This error often results in disaster. The harm done is due not only to delay in the administration of antitoxin but also to the fact that the patients are often subjected to incision of the peritonsillar tissues. Such procedures open up wide avenues for the spread of the infection and the absorption of the diphtheria toxin. Six patients, whose ages ranged between twenty-four and thirty-six and who had been subjected to peritonsillar incision, were observed over a five-year period. Three died of toxic myocarditis and other complications. Two of the surviving patients also showed evidence of toxic myocarditis. The high mortality rate and prolonged convalescence of the survivors are directly attributable to unnecessary surgical manipulation and delay in the administration of antitoxin. Whenever a patient with a supposed peritonsillar abscess is examined, diphtheria should be ruled out by repeated cultures. Incision of the peritonsillar tissues should not be done hastily. By following this conservative routine, they believe that the high mortality and the prolonged morbidity in this condition will be reduced.

True peritonsillar abscess (quinsy) in this part of the country is treated conservatively until localized fluctuation is present. Incision and drainage is then the procedure of choice. The results are excellent. Only rarely do we see a case of peritonsillar abscess in which the walled-off abscess forms at the base of the tonsil necessitating tonsillectomy for drainage. But Linck,[111, 112] Schroeder,[113] Thacker Neville[114] and Merica[115] advocate partial or complete tonsillar enucleation in the acute stage of peritonsillar abscess. Linck believes that there are often small collections of pus hidden away in the depths of the peritonsillar tissues which are drained by this procedure. With infrequent drainage such conditions may lead to thrombophlebitis of the large neck veins, an invasion of the blood stream and often a fatal ending. The local signs in the throat are few and may be

overlooked. He adds that abscess-tonsillectomy is the only certain way of opening and draining these dangerous hidden abscesses and that in peritonsillar abscess, primary abscess-tonsillectomy should be the operation of choice.

Schroeder, in presenting 56 cases, says that partial or total tonsillectomy, in order to get the pus as soon as possible, has yielded very good results, and that it is not proved that the procedure gives a less satisfactory postoperative course than incision. He states: "It is remarkable how many cases of peritonsillar inflammation are complicated by parapharyngeal abscess."

Merica has had no complications in 21 cases in which tonsillectomy was performed for quinsy and each patient showed immediate improvement.

In the ordinary case, however, this form of treatment seems to the reviewers more radical than necessary, and until it proves its merits should be left to the rare type of peritonsillar abscess which localizes at the tonsil base. It is to be emphasized, furthermore, that fatalities from unrecognized deep pus in the neck, secondary to tonsillitis and peritonsillar abscess, are continually occurring. The parapharyngeal or pharyngomaxillary space is the common site for accumulation of pus. Early adequate drainage must be done externally. The great vessels of the neck easily become involved. Boemer[116] emphasizes this point and Schroeder[117] describes the anatomic factors which explain the migration of pus in this area, outlining the surgical treatment of the condition involved. Infection from the tonsil occasionally spreads to the cavernous sinus by one or more of several routes. It may reach the internal jugular vein by the pharyngeal veins and then by the posterior route, passing by the inferior and superior petrosal sinuses to the cavernous sinus. On the other hand, it may pass from the jugular vein by the anterior route, through the facial, angular and ophthalmic veins to the sinus. A third route, he says, is by the pterygoid plexus and the vein through the foramen ovale to the sinus.

Tavani[118] reports a fatal case of cavernous sinus thrombosis following acute tonsillitis. Jauerneck[119] reports a case that recovered after operation on the ethmoids to relieve tension in the orbits; the sight of one eye was lost, however. Grove[120] reviews the literature on cavernous sinus thrombosis. His conclusions are as follows:

> There are septic and aseptic types of thrombosis of the cavernous sinus. The aseptic types are caused by penetrating injuries involving the sinus, by accidental injury to the sinus during an operation in its neighborhood and by fracture of the skull and injury to the head. In this group must also be included the marasmic types of thrombosis.
>
> In the septic types in which the thrombosis or thrombophlebitis reaches the cavernous sinus by way of its afferent vessels the mortality is practically 100 per cent.
>
> In those cases in which the condition is septic and of the restrained or chronic compensatory type, in which the thrombotic process reaches the cavernous sinus by retrograde action along its efferent vessels, there is some hope of recovery. In the cases of this group in which recovery occurred the thrombotic process in the cavernous sinus itself must have been aseptic, although originating from a septic source elsewhere.
>
> The percentage of recovery from all types of thrombosis of the cavernous sinus is probably not greater than 7 per cent. The percentage of recovery from the purely aseptic types is considerably higher than this.
>
> Operations on the cavernous sinus have been extremely disappointing and have shown poorer results than nonoperative treatment. When the thrombosis originates from a septic source the focus should be vigorously treated and eradicated. The cavernous sinus itself should be left alone. As an exception to this general statement, it would seem that ligation of the carotid artery, as proposed by Eagleton, might prove beneficial.

PHARYNX

After removal of the faucial tonsil there is a tendency in certain cases for the lateral pharyngeal bands to increase and to give rise to an unpleasant sensation. This may consist of sore throat, a sensation of a foreign body, or accumulation of mucus, causing swallowing or hawking. In such cases the topical application of remedies to the pharynx is disappointing. Lillie[121] has found that the ingestion of iodides gives good results, although he is unable to explain their action.

Schenck[122] studied histologic sections of tissue from the pharynges of 108 patients with systemic disease and chronic pharyngitis. These invariably presented evidence of hyperplasia of the lymph nodules and chronic inflammation. In these patients the histopathologic changes were common to all the types of disease studied; no specific histopathologic characteristic differentiated the pharyngeal nodules of patients with one type of disease from those of patients of the other groups.

Brown[123] emphasizes focal infection resulting from the chain of lymph follicles in the lateral pharyngeal wall. He thinks that involvement of the lymphoid follicles or glands of the pharynx is rarely if ever a primary focus of infection. As a secondary focus it is a definite factor and source for the distribution of infections, and has long been overlooked or unrecognized as such. He believes that the elimination of this focus aids in preventing infections which originate in the adenoids, tonsils, teeth and sinuses from being continued indefinitely.

Stout[124] reports the first case in the literature of a dry-ice (carbon-dioxide ice) burn of the throat. Recovery was painful but otherwise uneventful.

Retropharyngeal abscess, commonly regarded as a condition of no special surgical risk, still carries an average mortality of 7.4 per cent, according to Richards.[125] Only its constant consideration as a possible explanation for a wide range of symptoms

will prevent diagnostic errors. Careful digital palpation of the pharyngeal wall is preferable to the use of a tongue depressor or mouth gag; and pharyngeal incision, without anesthesia and in the prone position, will suffice to secure drainage in almost all cases. He adds that sudden severe hemorrhage must at once be controlled by carotid ligation. Schall[126] reports a case of an aberrant vessel of the posterior pharyngeal wall that ruptured four days postoperatively following evacuation of a hematoma which simulated a retropharyngeal abscess. Ligation of the common carotid artery stopped the bleeding, which was temporarily controlled by pressure in the nasopharynx and compression of the common carotid artery. Four months previously a retropharyngeal abscess in this location had been incised and drained.

Tumors of the pharynx, says Pilcher,[127] can be divided into two groups: (1) epilaryngeal, occurring almost exclusively in men; (2) postcricoid, seen almost exclusively in women and about ten years earlier than in men. In the epipharyngeal group, the commonest early symptom is a persistent abnormal sensation in the throat, varying in kind. Other early manifestations are enlarged nodes in the neck or interference with the voice. Postcricoid tumors are characterized by a long history of slight difficulty with deglutition; other symptoms are at first intermittent. A single serious attack of dysphasia in a patient with a long history of slight difficulty calls for a thorough examination. In the majority of cases, when symptoms first appear some abnormality can be seen by indirect laryngoscopy. These abnormalities are described. One hundred cases are analyzed; radical operations were completed in 39 patients, with the death of 16 and a survival of 7 for periods of from eighteen months to ten years. He makes a plea for closer investigation of minor disturbances of the voice and of swallowing, with the use of indirect laryngoscopy in suspicious cases.

Albright[128] reports the case of a forty-nine-year-old man with a large tumor on the right side of the neck, of seven years' duration. The growth protruded into the oropharynx, displacing the uvula upward and to the left. The overlying mucosa was intact. The firm, encapsulated growth, 8 cm. in diameter, was removed through a longitudinal incision in the neck. Histologic study showed it to be a fibroma, probably of perineural origin. The patient has remained well for two months after operation.

Torres and Monteiro[129] report a case of an unoperated upon retropharyngeal neurinoma in a man fifty years of age. The tumor, which had been growing slowly for about six years and measured some 6.5 by 4.5 cm., completely filled the retropharyngeal space. At autopsy a number of large cavities filled with pus were found; during life, arteriography and exploratory punctures had shown these spaces to be filled with blood. There was no metastasis.

Kanas[130] describes the case of a man forty-seven years of age with destructive ulceration which began on the posterior surface of the soft palate, and in a few weeks spread so as to involve the whole of the nasopharynx and a part of the pharynx. Secondary infection of the extensive, gangrenous, ulcerated surface was severe, and hemolytic staphylococci were found both locally and in blood cultures. No treatment was of any avail, and in about three and a half months from the beginning of the illness death followed hemorrhage from an eroded vessel. Microscopic examination of several specimens, removed during life, showed only ordinary granulation tissue. Further examination after death, however, showed that the disease was in reality an atypical lymphosarcoma.

In the Semon Lecture for 1936, Howarth[131] described cases of tumors and ulcerations of the palate and fauces. He outlined the technic of diathermy excision of malignant tumors which he has used during the last twenty years, and gave his results in 61 cases. After a brief review of the various forms of ulceration that are found in this region, he described a form of ulceration under the name of precancerous epithelioma and gave the histories of 3 cases.

NASOPHARYNX

Hill[132] stresses the fact that in malignant disease of the sinuses and nasopharynx certain common errors are encountered. One of these is failure to make a correct diagnosis through overlooking the nasopharynx as a possible site of the primary lesion, or through neglecting to have proper roentgenologic and pathological studies made. Another is a misguided zeal to preserve the cosmetic result at the expense of being sufficiently radical. Fortunately, both these faults may be easily remedied.

Flynn[133] reports a case of fibromyxosarcoma of the nasopharynx in a boy of twelve. This was removed and the boy has been well for nine years without recurrence.

A case of plasma cell tumor of the nose and nasopharynx is reported by Blumenfeld.[134] According to the author, who reports the abstracts of previous cases, this is the twenty-first case presented in the literature.

CYLINDROMAS

The name "cylindroma" is given to a numerous variety of tumors which differ in their clinical development, in the type of therapy required, and in their prognosis. The term ought to be abandoned, say Lemaitre, Ardoin and Lemaitre.[135] Tu-

mors exist which are histologically cylindromatous but have few characteristics suggesting a common origin. Some are undoubtedly malignant tumors; on the other hand, others are relatively benign.

They add that the cylindromatous epitheliomas arise from seromucous glands, which are chiefly found in the upper digestive and respiratory tracts, the mouth, the pharynx (above all, the nasopharynx), the larynx and the trachea. They remain submucous for a long time, do not ulcerate, are slow-growing, show very little lymphatic spread, and never metastasize. They call for surgical removal, but often recur several months or years after operation, generally ending in death from seven to ten years later after three or four operative interventions; their degree of radiosensitivity has not yet been established. The cylindromatous precancerous states are commoner than the true cylindromatous epitheliomas, and their history is confused with that of the so-called "mixed tumors" of the salivary glands. Precancerous tumors are found, in order of frequency, in the parotid region, the arches and velum of the palate, and the submaxillary glands. Their development is slow and they have a good prognosis if completely removed, but their transformation into cancers is far from exceptional, and in this respect they differ from the mixed tumors. The cylindromatous epitheliomas of the skin resemble classical cutaneous epitheliomas, especially the basal-celled variety. Clinically, good results follow rational treatment — total excision.

Goldsmith and Ireland[136] report 6 cases of aberrant mixed tumors of salivary gland type. The general consensus is that these tumors are not true teratomas. Those closely associated with the glands proper probably arise from the gland ducts, and the aberrant type from embryonal rests. Cartilage and myxomatous tissue can be developed by metaplasia, and their mesodermal origin is not considered essential. Tumors of this type involving the accessory sinuses are rare. One such case has been reported. The treatment by complete surgical removal was the most satisfactory measure in their series. Radiation as a primary treatment should not be considered; prophylactic postoperative radiation may have some usefulness. They add that recurrence of the growth is frequent, and make no attempt to report any of the cases as cures.

LARYNX

Tucker[137] reports an unusual congenital case of ptosis of the larynx in a child, due to replacement of the left sternohyoid and sternothyroid muscles by a tendinous band pulling the hyoid bone and the larynx downward. The result of the resection of the fibrous band has been satisfactory in overcoming the displacement. The child had a speech defect partly caused by interference with the proper use of the tongue by the displacement of the hyoid bone. Correction of the abnormality improved the speech.

Central lesions that produce paralysis of the larynx are discussed by Furstenberg.[138] Corticobulbar fibers carry motor impulses to the lower motor neurons which supply the larynx. Attention is first called to the pontine division of the aberrant pyramidal tract and the partial decussation of the corticobulbar fibers for the laryngeal group before they terminate in the nucleus ambiguus. Impulses brought to the recurrent nerves therefore arise in both the right and left precentral regions of the cerebrum, an anatomic fact of much importance when one is endeavoring to localize lesions causing laryngeal paralysis of central origin. Secondly, lesions above the nucleus ambiguus — the origin of the laryngeal nerves — cannot produce a unilateral paralysis of the larynx on account of the decussation of the corticobulbar fibers. When the lesion is large enough to involve them on both sides, a bilateral paralysis of a spastic type will be observed. Attention is called to a differentiation seldom made by the clinician, namely the spastic versus the flaccid type of laryngeal paralysis. Supranuclear lesions produce the former, nuclear and infranuclear lesions the latter. Thirdly, unilateral laryngeal paralysis of central origin is a not uncommon condition. It is seldom observed, however, because it is usually associated with a preponderance of obvious neurologic findings, which invite study and minimize the attention given to the special organs of the body. He adds that, in many cases of this kind, the symptoms are so trivial that neither the patient nor the doctor is aware of their presence.

Lore[139] and Sercer[140] present operative procedures for the relief of laryngeal stenosis due to bilateral abductor paralysis.

Hofer and Motloch[141] point out that membranous deformities of the vocal cords occur chiefly on the anterior commissure but occasionally on the posterior commissure of the cords. They vary in size from a slight web formation to complete atresia of the lumen. The genesis of these membranes has not been completely explained, but some authors regard them as embryonal remnants. They believe that the deformities are not necessarily congenital, for there are a number of disorders that may cause the formation of cicatricial membranes in the region of the glottis, namely syphilis, diphtheria, tuberculosis, typhoid perichondritis, leprosy, rhinoscleroma and glanders. These authors report a case in which tracheotomy and a bolt pushed up into the anterior commissure were necessary to obtain permanent cure. They state

that chronic inflammatory tumors of the true vocal cords tend to increase in size because of the functional activities of the cords.

According to Tucker,[142] a vicious circle is set up in which local irritation, increase in the size of the tumor, and the overacting musculature of the larynx, all became important etiologic factors. Restoration of the normal contour of the cord by direct laryngoscopic removal of the tumors and by voice training to restore the normal muscular action will eliminate the vicious circle and restore the larynx to normal in most cases. The microscopic examination of the tissue removed by direct laryngoscopy provides a histologic basis for diagnosis.

Koch[143] gives the clinical histories of 22 cases of papilloma of the larynx treated by x-ray, and describes the exact dosage and methods of irradiation.

Palmer and Mehler[144] report a case of recurrent lipomyxochondrofibroma of the larynx operated on five times in five years.

Cummings[145] reports a case of lymphosarcoma of the larynx associated with skin manifestations and later with generalized fatal metastases.

Clerf and Bucher[146] write on blastomycosis of the larynx. Their conclusion is that in extensive inflammatory lesions of the organ with ulcerative and exudative changes which, although suggestive of tuberculosis, are not characteristic of any definite clinical entity, one should consider fungus infection as an etiologic possibility. In the 3 cases cited a tentative diagnosis of tuberculosis had been made. Repeated negative sputum studies for tubercle bacilli and negative pulmonary findings in spite of histologic diagnoses of chronic ulcerative laryngitis either granulomatous or suggestive of tuberculosis, indicated a need for additional investigation. The final diagnosis rested on the results of the mycologic findings, together with the above-noted findings. Fungi were demonstrated in all the lesions. They produced definite lesions in rabbits. The positive mycologic findings at repeated studies and the improvement under iodide therapy were conclusive. In 2 other cases the findings were suggestive from a clinical standpoint. Although fungi were found in both cases, their pathogenicity was inconclusive.

A number of papers have appeared on tuberculosis of the larynx.

Schuster[147] studied the larynx from the clinical standpoint in 452 cases of pulmonary tuberculosis with subjective symptoms referable to the ear, nose or throat. Tuberculous laryngitis was present in 192; diseases of the nose and throat were no more frequent in these patients than in those in whom the disease was limited to the lungs. In this group only age, sex and pregnancy had any bearing on the development of tuberculous laryngitis. Tuberculous infection in the larynx usually comes from contact with bacteria-laden sputum, although it is probable that some cases, in which the sputum contains no bacilli, are lymphatic in origin. A strong local resistance to clinical invasion by the tubercle bacillus was noted in the upper respiratory tract, but no such immunity was observed for other infections.

He continues that reflected sunlight is occasionally of value in supervised cases. When the lesion is progressive, the radical and repeated use of the galvanocautery is of great worth, care always being taken not to exhaust the patient at any one sitting. Time and temporary relief may be gained by blocking the superior laryngeal nerve. The actual cautery is now well recognized as a most valuable aid, and he believes that its use has been too frequently reserved for patients with far-advanced and more or less hopeless conditions. Early use is urged, especially when the condition does not respond promptly to conservative treatment. He adds that the pathologic picture is essentially that of tuberculosis elsewhere and is extremely protean and that the diagnosis should be based on the gross observations in the larynx, with the confirmatory observations in the chest and the results of laboratory and roentgen study. Prognosis as to healing of the laryngeal lesion in the early stages is good, but uncertain as to ultimate recovery. Early treatment is based on co-operation in general care with the specialist in diseases of the lungs, on absolute rest of the voice, on pulmonary collapse in suitable cases and on avoiding overtreatment locally.

Blegvad[148] takes up the problem of early laryngeal tuberculosis. His article is based on 1773 cases suffering from laryngeal tuberculosis that were treated in Copenhagen from 1916 to 1934. In 404 cases the final result is unknown, but of the remaining 1369 patients 74 per cent have died and 26 per cent are alive and well. The treatment has been that inaugurated by the Finsen Institute, namely universal carbon-arc-light baths or quartz-light baths. The patients are all ordered to keep silent. If there is no appreciable improvement within a few months, a local operation is carried out if the condition of the patient permits; it may consist of one or more of the following: galvanocauterization, amputation of the epiglottis, excision of the affected part, injection of alcohol and resection of the superior laryngeal nerve. The author stresses the importance of early diagnosis, and emphasizes the following clinical diagnostic points: (1) isolated redness of the vocal cord; (2) swelling and redness of the vocal process; (3) prolapse of the ventricle of Morgagni; (4) swelling of the lower surfaces of the vocal cords; (5) swelling

of the mucous membrane in the interarytenoid region; and (6) a red cushion beneath the commissure.

And we must not forget, as Hautant[140] emphasizes, that cancer may simulate tuberculosis and vice versa. Laryngeal tuberculosis can take the aspect of a warty, subglottic and unilateral lesion. Sometimes it resembles a ventricular tumor. In both instances the clinical aspect is that of an intralaryngeal epithelioma; a very careful examination is necessary to avoid an error. A radiograph of the larynx may be a valuable aid in the diagnosis of cancer. In doubtful cases, several biopsies should be carried out from different parts of the laryngeal lesion. Deep roentgenotherapy, even as a test treatment, should be resorted to only with great caution. He states that, even in cases in which a diagnosis of epithelioma seems obvious, a pulmonary examination and x-ray should always be made and taken into consideration.

Rubaltelli[150] reviews the literature and states that the results obtained with either periarterial sympathectomy or roentgen treatment in tuberculous laryngitis are identical. Both treatments control mechanically painful dysphagia and produce reflex intralaryngeal vasodilatation. The results reported in the literature concerning the effects of both treatments on infiltration or ulceration of the laryngeal mucosa are conflicting. He prefers the roentgen treatment, since it can be administered in all cases, regardless of the local condition of the disease and of the general condition of the patient. If it is given in the precise dose and with the proper technic, accidents and complications do not follow. Furthermore, it is advisable to avoid, whenever possible, bleeding and prolonged or complicated operations in tuberculous patients. The roentgen treatment can be given as an ambulatory treatment and repeated at certain intervals. He believes that the benefits of repeated roentgen therapy are more lasting than those obtained from sympathectomy.

Safranek[151] describes x-ray treatment in patients with tuberculous laryngitis, and gives the dosage. He has treated 300 cases in all, and in infiltrative or "productive" cases obtained a cure in 70 per cent. X-rays were not used in advanced cases of the exudative type, but they have been found helpful in relieving dysphagia. He concludes that x-rays have proved to be of great service in the treatment of laryngeal tuberculosis.

Eschweiler[152] finds carbon-dioxide snow useful in early types of tuberculosis of mucous membranes.

The surgical treatment of carcinoma of the larynx continues to make progress, and may now be regarded as the most satisfactory method of treatment. Looper[153] states that in a series of 35 cases no serious operative complications or fatalities have occurred. The convalescence has been satisfactorily rapid and pleasing. These patients are not depressed, and most of them are made happy and given a new lease on life with the aid of the artificial larynx.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23441

PRESENTATION OF CASE

A sixty-three-year-old white female was admitted complaining of recurrent, severe attacks of epigastric pain.

Four and a half months before entry she began having severe attacks of epigastric or substernal pain which felt like "gas" or "acid." She had no true smothering or constricting sensations. The pain was referred to the forearms, but not to the upper arms or hands. The attacks lasted half an hour or more, occurred four or five times a week or as often as two or three times a day, were not brought on by exertion, and were more apt to occur while lying quietly. Many of the attacks required morphine for relief. Nitroglycerin had no effect.

Physical examination showed a well-developed and nourished elderly woman lying in bed in no discomfort. The heart was not enlarged and the rhythm was regular. There was a rough systolic apical murmur. The blood pressure was 116 systolic, 72 diastolic. There was definite tenderness in the right upper quadrant. The left wrist and the intercarpal-phalangeal joints of the first two fingers of the left hand were somewhat enlarged and tender. There was some deformity of the joints of all the fingers, and there was some limitation of rotation of the hips. The physical examination was otherwise essentially negative.

The patient had spent two periods in this hospital, eight and nine years previously, with a diagnosis of acute bursitis of the left shoulder. X-rays of the shoulder, gall bladder and gastrointestinal tract were negative. One and a half years ago she spent two months in this hospital with a diagnosis of rheumatoid arthritis of the hands, spine, hips and knees. The past history was otherwise essentially negative.

The temperature was 98°F., the pulse 70. The respirations were 20.

A urine examination showed the slightest possible trace of albumin. The red cell count of the blood was 3,800,000; the white cell count was 7,800, 60 per cent polymorphonuclears. The nonprotein nitrogen of the blood serum was 16 mg. per cent. Guaiac tests of the stools were repeatedly negative. A blood Hinton test was negative. An electrocardiogram showed normal rhythm, a PR interval of 0.16 sec., normal T waves and moderate left axis deviation.

X-ray examination showed a small hiatus hernia but otherwise normal esophagus, stomach and duodenum. The vertebrae, chest and diaphragm were also normal. A Graham test showed a normal functioning gall bladder. An x-ray of the chest was negative.

On the day following admission she had two attacks of severe substernal discomfort which she did not describe as true pain but rather as "agony." During the attacks her pulse was regular with a rate of from 110 to 120. Both attacks were relieved by morphine, which the patient demanded. During the attack she had eructations of gas, which gave her some relief. For the next two weeks her condition remained essentially unchanged. She had about two attacks a day, all of which were similar, and many of them required morphine for relief. During an attack on the thirteenth hospital day she was given 10 cc. of calcium chloride intravenously, which apparently relieved her chest pain very promptly. A white blood-cell count the next day was 13,700, 56 per cent polymorphonuclears. On the eighteenth day the patient said that for the previous week she had had an "empty, all-gone feeling" in the pit of her stomach and described a similar discomfort which she had experienced twenty years previously at the time of the death of her brother-in-law. She said that telling about this incident would probably bring on an attack of substernal "agony," and an attack immediately supervened which was not relieved by prolonged inhalation of amyl nitrite. There was no change in the pulse except for acceleration of the rate. The following day the patient appeared mildly depressed and talked about going home and not eating until she died. A neurologic examination on the twenty-third day was essentially negative except for atrophy of the first interosseus muscles of both hands. The following day the blood pressure was 100 systolic, 80 diastolic. Early in the morning of the twenty-sixth day she suddenly experienced very severe pain in the chest which radiated to both arms. She was somewhat dyspneic and perspired freely, and the skin was cold and clammy. There were rales throughout both lungs. The heart rate was 100, and the blood pressure was 70 systolic, ? diastolic. She was very restless, thrashed about in bed, and had to be restrained. Oxygen administration improved her color, but her pulse gradually weakened. An emergency electrocardiogram showed marked changes in the T waves and QRS complexes. T_1 was diphasic, and T_2, T_3 and T_4 were upright. The QRS complex was inverted, and there was intraventricular block. The PR interval was 0.22

sec. She died on that day, twelve hours after the onset of the attack.

Differential Diagnosis

Dr. William B. Breed: I gather from the history that the physician in charge of this patient was in a quandary, and I think he found himself in the same position that many of us have been in when treating someone whom we suspect of being a marked psychoneurotic until death supervenes. You will note particularly that there are various statements emphasized to indicate that she was of a psychoneurotic personality; such as, "the attacks never came on with exertion and were more apt to occur while lying quietly . . . nitroglycerin had no effect . . . the attacks were relieved by morphine, which the patient demanded." There is also an addiction inference here. Then she was given 10 cc. of calcium chloride, which was probably an attempt to show that this pain was not coronary in origin but perhaps due to smooth muscle contraction in the esophagus, stomach or gallbladder region. I am not sure how much work has been done with calcium chloride in the relief of anginal pain. Dr. White, is that a good therapeutic test?

Dr. Paul D. White: I know nothing about it.

Dr. Breed: Does the fact that this pain was relieved by calcium chloride tend to support the absence of coronary disease or coronary artery contraction? (No answer.) I think it was probably done with that in mind.

She had substernal "agony," and an attack occurred as predicted. She had some feeling of impending death and the fact remains that she *did* die, but *not* of psychoneurosis. It is very much easier for me knowing that she died, and having her record before me, to go back and look for facts in the history which substantiate a diagnosis of organic disease. The man who was in charge of this case did not have the advantage we have in evaluating this patient's condition.

I am interested in one statement in the second paragraph where it says that the pain was referred to the forearm, but did not radiate to the upper arms or hands. It was not a radiating pain from the chest but just occurred spontaneously in the forearm. I have seen that in one case of coronary disease preceding coronary occlusion. It was the most striking part of that man's history — isolated pain without radiation. I am putting a good deal of emphasis on that statement.

She had no cardiac enlargement and no hypertension that we know of. We cannot consider 116 mm. hypotension unless we know what it was before. She had rheumatoid arthritis undoubtedly, but I do not think it needs to come into the picture. There is no evidence of disease below the diaphragm, either by history or examination.

There is one other interesting statement. Nine years before entry she had acute bursitis of the left shoulder. We must scan that a little more closely. It might have been anginal pain. I have seen one patient die with massive coronary occlusion who several months before had been given a tentative diagnosis of bursitis of the left elbow. It was never confirmed, but he did have pain in the left elbow region that suggested a bursa. There was no evidence by x-ray of calcium deposit, and it may not have been a true bursitis.

The electrocardiogram is essentially negative at entrance, which is nearly a month before death, except for left axis deviation. The x-ray helps not at all except in excluding subdiaphragmatic lesions. During the last episode she suffered severe shock. That in itself does not necessarily mean that she died of coronary occlusion because similar shock can be produced by the passage of a gallstone through the common duct or by any severe pain. The fact that she was very restless and thrashing around in bed is by and large against coronary occlusion because most of the people who are suffering from pain of coronary occlusion like to be quiet. On the other hand, there are some patients who have such agonizing pain that they cannot stay still. The drop in blood pressure *in itself* does not mean coronary disease. It may have been other pain that caused the reduction of systolic pressure from 116 to 70 in a short time. However, it is all consistent with the picture of cardiac infarct. The electrocardiogram brings up the question as to whether this was done before the change in technic of recording T_4.

Dr. F. Dennette Adams: It is the new T_4.

Dr. Breed: If this is the new T_4 then the upright curve must be interpreted as a normal one. Prior to the change in technic an upright T_4 would have indicated definite coronary disturbance. The emergency electrocardiogram is also not very abnormal except for a diphasic T_1. On the other hand, the report says that the T waves showed a *marked change*. I should like to know more about that. Dr. Sprague, will you be good enough to tell me about marked changes in the T waves, because this is a very important point.

Dr. Howard B. Sprague: The first electrocardiogram shows a moderate degree of left axis deviation, a moderately late inversion of the T waves in the third lead and a diphasic T wave in the second lead. As a matter of fact the third lead seems to be almost entirely inverted, as if it might be due to a high diaphragm, and the fourth lead shows a low, somewhat diphasic but mostly upright T wave. With the change in the technic it would be within normal limits.

Dr. Breed: How can we interpret it? Left axis

deviation, with change only in T waves in the third lead, a diphasic T wave in the second lead. We have to know whether she had had digitalis.

DR. ADAMS: I cannot remember.

DR. BREED: Let us assume that she did not have digitalis.

DR. SPRAGUE: We will say that she has not had digitalis, and the changes here in the first electrocardiogram are not pathognomonic of disease in the coronary vessels.

DR. BREED: Then what would you say? Is the electrocardiogram normal?

DR. SPRAGUE: It looks to me as if the changes might be found with a hypertensive heart.

DR. BREED: Is there any indication of coronary disturbance?

DR. SPRAGUE: I cannot make a diagnosis of coronary thrombosis, but, again, such changes would not be inconsistent with it.

DR. BREED: But she might have some coronary disease without having any coronary occlusion? She might on that record have had coronary disease?

DR. SPRAGUE: That is right.

DR. WHITE: Do you interpret the murmur as of any significance? In spite of the normal size of the heart that would mean she might have enough involvement of the heart to cause a little involvement of the left ventricle.

DR. BREED: I was inclined to disregard it. She may have had enlargement of the ventricle to give left axis deviation, but I did not interpret the murmur as meaning anything.

DR. WHITE: It was a rather prominent one.

DR. SPRAGUE: On the afternoon of the day of the first electrocardiogram, a second was taken; this was essentially the same, except for a little displacement of the ST interval in the fourth lead, which could be consistent with coronary disease. The last one taken shows a marked change. The change, however, is in the conduction within the ventricle, intraventricular block, and apparently some disturbance in conduction between auricles and ventricles. There seems to be a partial AV block.

DR. BREED: The PR interval is increased at least 0.06 sec.

DR. SPRAGUE: Yes, and the conduction time within the ventricle is increased. You have development of intraventricular block, which would indicate myocardial change; but the T-wave changes are not diagnostic so far as coronary thrombosis is concerned.

DR. BREED: Are the changes in the T waves marked as it says here, and if they are what does it mean?

DR. SPRAGUE: They are marked because changes in the whole ventricular complex are marked. Development of intraventricular block has changed both QRS and T.

DR. BREED: How long before death was it taken?

DR. ADAMS: It was eight hours before death; about two hours after the onset of the final episode.

DR. BREED: Of course an electrocardiogram taken two hours after a bad coronary occlusion is not expected to show any marked changes, but I am going to be orthodox and put all my chips on the diagnosis of coronary disease, with a coronary occlusion at the end. I think it is quite unlikely that she had dissecting aneurysm or pulmonary embolus. This case illustrates two important points. The first is the great difficulty a person gets into when the diagnosis of psychoneurosis is assumed and when definite evidence to back up an organic diagnosis is lacking. Another point is that it is quite possible that we might have seen more evidence indicating coronary disease if we had had serial examinations by electrocardiogram during her whole stay. We know that people with coronary disease may at times show disturbance in conduction by electrocardiogram on one date and at another time have a normal one. I think that we might have been able to make a more definite diagnosis of heart disease with repeated serial electrocardiograms. I shall put as my diagnosis, coronary occlusion; the others that we have discussed in passing are possibilities but seem less likely.

CLINICAL DISCUSSION

DR. ADAMS: This woman was my patient. She lived in another city so that until she came to the hospital I did not see her in an attack. The first episode which occurred in February was described to me over the telephone, and from the description, coronary occlusion seemed most likely. However, a series of electrocardiograms taken within the next few weeks showed no appreciable variation from the normal, and further attacks of pain which occurred during March, April and May were not at all conclusive.

She was admitted to the hospital for further investigation because of the increasing severity and frequency of the episodes. We tried to convince ourselves that the attacks were due to coronary disease, but although she was observed in many of them, we were unable to do so. None of them appeared typical. Even leading questions could not bring out a description of anything suggesting compression, tightness, squeezing, oppression, breathlessness or a feeling of smothering. She would only describe her sensation as one of "agony." During each attack she became intensely hysterical, screaming, yelling and belching prodigiously. The attacks were never precipitated by

exertion. For diagnostic purposes she was permitted to walk up and down the corridor of the hospital on many occasions in the hope of precipitating an attack, yet one never started after this exertion. Her electrocardiogram did not show the usual changes.

The pain in the forearm which one would naturally consider as being of coronary origin rather led us away from such a diagnosis. This was always described as occurring in symmetrical areas on the two forearms, each area being no larger than a half dollar. Furthermore, on no occasion was any benefit received from nitroglycerin or amyl nitrite.

All the other studies which were done were carried out in an effort to introduce some other factor as a cause of the pain, because we did not feel that there was sufficient evidence to justify the diagnosis of coronary disease. Calcium chloride was tried in an attempt to exclude gall-stone or kidney-stone colic.

Until the onset of the final episode we were completely baffled and quite unable to make a diagnosis. We knew that we were dealing with a patient with marked psychoneurotic tendencies, but every man who saw her felt sure that there must be some organic background for the discomfort. I personally thought in terms of a possible dissecting aneurysm, although this was merely a guess.

The final episode which began the night before death was quite characteristic and at this time we felt that we were definitely dealing with coronary disease.

Dr. White: May I add a word about the electrocardiogram? These changes are considerable and probably not explained by her moribund state, but we must remember that terminal electrocardiograms may be misleading. On the basis of a terminal change a diagnosis of coronary thrombosis was made in a certain case by several observers; at autopsy that patient presented a normal heart and a gall bladder full of pus. The final electrocardiogram was taken a few hours before death when the patient was extremely ill and in a toxic state.

Clinical Diagnoses

Coronary occlusion?
Dissecting aneurysm?

Dr. Breed's Diagnoses

Coronary occlusion.
Rheumatoid arthritis.

Anatomic Diagnoses

Arteriosclerosis, marked aortic and coronary.
Coronary thrombosis, fresh, right main branch; old, left descending and circumflex.
Cardiac infarction, left ventricle.
Mural thrombus, left ventricle.
Myocardial fibrosis, diffuse.
Mural thrombus, abdominal aorta.
Operative scar: appendectomy.
Melanosis coli.
Multiple diverticula of colon.
Hydrothorax, bilateral.

Pathological Discussion

Dr. Tracy B. Mallory: This patient had a normal-sized heart without any hypertrophy of either ventricle. All three coronary arteries were completely occluded. The descending and circumflex branches on the left showed old lesions, and the right main coronary showed a fresh thrombosis. There was a fairly large area of old scarred infarction on the lateral surface of the apex of the left ventricle, undoubtedly the result of one of the first two of her episodes of coronary occlusion.

CASE 23442

Presentation of Case

A fifty-three-year-old Italian laborer was admitted complaining of cough and sore throat.

The patient had been well until four weeks before entry, at which time he caught a severe cold following exposure to a draft. The cold was associated with a sore throat, general malaise and a slight, nonproductive cough. The symptoms continued, and a week later the patient was compelled to give up work, and during the succeeding period led a bed and chair existence. Two weeks preceding his entry the cough and sore throat subsided slightly, but at this time he developed a vague pain across the lower anterior portion of his chest. The pain was steady, aching in character, and was of equal severity on both sides. It was not influenced by respiration, coughing or activity. There was also a slight ache in the back of the neck radiating across the shoulders. Shortly before coming to the hospital there was an exacerbation of the sore throat and considerable pain on swallowing. Associated with this there was tender swelling of the glands on both sides of the neck, more evident on the right side. There was no respiratory difficulty, but the patient noticed that his voice was "stuffy."

Physical examination showed a well-developed and nourished, rather pale, middle-aged man, who appeared to be acutely ill. The teeth were dirty in appearance, and there was moderate pyorrhea. The cervical lymph nodes were slightly enlarged. The heart was normal in size. The rhythm was regular. The blood pressure was not recorded. At

the right base posteriorly there were dulness, increased tactile fremitus and suppressed breath sounds. No rales were heard. The abdomen was soft. The edge of the spleen was felt two fingers beneath the costal margin. Rectal examination was negative.

The temperature was 101°F., the pulse 100. The respirations were 25.

Examination of the urine showed a specific gravity of 1.018, with a slight trace of albumin. The sediment contained an occasional white blood cell and occasional hyaline and finely granular casts. The blood showed a red cell count of 3,900,000, with a hemoglobin of 70 per cent. The white cell count was 51,100, and a differential count of a supravital preparation showed 9 per cent neutrophils, 4.5 per cent myelocytes, 13.5 per cent lymphocytes, 10.5 per cent monoblasts, 60 per cent monocytes, 2 per cent eosinophils, 0.5 per cent basophils and 1 per cent multinucleated giant cells. The platelets were diminished in number and small in size. An occasional nucleated red blood cell was seen. The reticulocyte count was 1.3 per cent. Examination of the stools was negative. A blood Hinton test was negative. A throat culture showed many colonies of hemolytic streptococci. The sputum on culture contained no pneumococci, but many colonies of *Staphylococcus aureus* and a moderate number of hemolytic streptococci. The nonprotein nitrogen of the blood serum was 23 mg. per cent. The serum chlorides were equivalent to 100 mg. N/10 sodium chloride. Blood cultures were negative.

An x-ray film of the chest showed the diaphragm to be high in position, but excursions were normal. There were haziness in the left costophrenic angle and slight mottling in the adjacent lung field, but the lung fields were otherwise clear. The heart was in the tranverse position and probably slightly enlarged. The aorta was not remarkable.

The patient's temperature rose to 105°F. and throughout the hospital course remained elevated between 102° and 106°F. A week after entry another portable x-ray film of the chest showed a slight increase in the amount of fluid in the left pleural cavity and what appeared to be a small area of consolidation adjacent to it. There was homogeneous dulness in the right upper lung field, said to be consistent with pneumonia of the right upper lobe. On the eighteenth hospital day the patient complained of pain in the right ear, and subsequent examinations revealed otitis media on this side. Paracentesis of the right drum was done, but despite drainage the patient's temperature remained markedly elevated. The red cell count of the blood gradually diminished to 2,050,000, with a hemoglobin of 50 per cent, and the white cell count to 15,000, although the differential formula was essentially unchanged. Despite transfusion and other supportive treatment the patient's condition became rapidly worse; he lapsed into coma and died on the twenty-ninth hospital day.

Differential Diagnosis

Dr. Bernard M. Jacobson: This patient entered the hospital with a story which was indistinguishable from that of an ordinary acute respiratory infection. There is nothing in the physical findings that is against this presumptive diagnosis. The signs at the right base posteriorly might have been due to atelectasis of the right lower lobe. The finding of a palpable spleen is by itself not enlightening. It is to be noted that only slight enlargement of the cervical lymph nodes was found. Apparently there was no other significant adenopathy, and the liver is not commented upon. The first significant finding that is not in agreement with a diagnosis of respiratory infection alone is that of the white blood cells. The total white blood-cell count and differential picture are certainly not compatible with an ordinary respiratory infection, and are more consistent with a primary disturbance of the hematopoietic system.

In the history it was stated that the illness was ushered in with a sore throat. We do not know whether at that time the throat was merely inflamed or whether in addition it was severely ulcerated. Sore throat as an early symptom is often seen in leukemia, infectious mononucleosis, agranulocytosis, purpura hemorrhagica and aplastic anemia. All these conditions but one are difficult to fit in with the rest of the picture. The age of the patient and the high percentage of monoblasts are definitely against a diagnosis of infectious mononucleosis. The high total leukocyte count and the presence of 4.5 per cent myelocytes rule out agranulocytosis. The absence of petechiae and the definite presence of blood platelets in the smear make purpura hemorrhagica unlikely. The high total-leukocyte count is definitely against aplastic anemia. On the other hand, the presence of 60 per cent mature monocytes and 10.5 per cent monoblasts is perfectly characteristic of monocytic leukemia. The duration of the illness, about four weeks, characterizes the leukemia as of the acute type. The fact that the total white cell count decreased during the hospital stay to 15,000 is not at all uncommon in cases of acute leukemia.

Clinical Discussion

Dr. Edward A. Gall: As Dr. Jacobson has said, the diagnosis in this case is clear cut. A relatively rapidly fatal process, initiated apparently by nasopharyngeal infection and pursuing a marked febrile course, is a clinical feature which, linked with a

moderately enlarged spleen, little or no general lymph node enlargement, and a subleukemic leukocytosis, points very strongly to an acute leukemia. Conclusive diagnosis, however, is impossible without knowledge of the morphologic structure of the blood elements.

The fixed Wright-stained smear in this case was extremely interesting in that the granular content of the predominating monocytes was unusually prominent. Under ordinary circumstances monocytes exhibit a rather fine cytoplasmic stippling with azurophilic granules. Coarsening of the granules and variation in staining characteristics prevent exact differentiation of these cells from myelocytes. Such was the initial clinical error in this case. The difficulty was promptly obviated, however, by supravital studies.

A moist blood film stained with neutral red and Janus green showed readily recognizable cells of the monocytic series, and these were easily distinguished from the few myelocytes accompanying them. It is highly probable that the similarity of these cells in the fixed smear accounts for the contention of certain authorities that monocytic and myelogenous leukemia are different manifestations of the same disease. They are not, of course, at all similar histologically.

The myelocytes in the blood probably represented simply a displacement phenomenon resultant upon bone marrow infiltration by neoplastic tissue.

Clinical Diagnoses

Monocytic leukemia.
Bronchopneumonia.
Acute mastoiditis, right.

Dr. Jacobson's Diagnoses

Acute monocytic leukemia.
Bronchopneumonia.
Otitis media, right.

Anatomic Diagnoses

Monocytic leukemia, acute.
Bacterial endocarditis, acute: mitral and aortic.
Pulmonary infarct, right upper lobe.
Perisplenitis, acute fibrinous.
Pleuritis, chronic fibrous: bilateral, focal.
Bronchopneumonia.
Splenic infarction, multiple.
Renal infarction, multiple.

Pathological Discussion

Dr. Tracy B. Mallory: The recognition of monocytic leukemia as a distinct and by no means rare clinical entity has occurred only within the past few years in most hospitals in this country. The diagnosis can, of course, only be made by careful examination of the blood. In a case such as the present one a high percentage of the cells are fairly mature monocytes and readily recognizable in the routine blood smear. In other cases, however, the majority of the cells may be quite immature and only the supravital technic will permit their recognition. In any acute leukemia such a study should never be omitted.

Since our recognition of the disease is still rather recent we know less about its behavior than about that of the other, commoner forms of leukemia. Clinically it tends to be relatively acute, and perhaps for that reason leukemic infiltration of the viscera is generally less marked. In this case the spleen and liver were both considerably enlarged, the former weighing 1150 gm. and the latter 3150 gm. There was a generalized lymph-node enlargement, though no gland more than 2 cm. in diameter was found; and the bone marrow was diffusely involved. We found no infiltration of the heart, kidneys, intestinal tract or skin. In the microscopic sections of the involved tissues the infiltrating monocytes were, like those in the peripheral blood, rather exceptionally well preserved. In two other cases which we have presented at these conferences we have commented upon the presence in the spleen and lymph nodes of large numbers of multinucleated cells very suggestive of Sternberg cells in their appearance. Doan and Wiseman,* also, in their excellent paper on the disease, note that the picture in the spleen and lymph nodes may be strongly suggestive of Hodgkin's disease. In the present case, such multinucleated cells, though present, are quite infrequent and only one is found in every four or five high-power fields.

Grossly the spleen showed many hemorrhagic infarcts varying from 1 to 4 cm. in diameter. Histologically it seemed clear that these really consisted of multiple, closely packed, miliary infarcts. Even in the grossly noninfarcted portions, microscopic examination showed many minute infarcts in all stages of organization, and a considerable increase in fibrous tissue seems to have been brought about in this manner.

A clinically unsuspected finding at autopsy was an acute endocarditis of the aortic and mitral valves. Whether this was due to *Bacillus proteus,* which grew in abundance from the blood stream, seems impossible to prove. Infarcts were present in the kidneys, and in retrospect one naturally wonders whether it was the leukemia or the endocarditis which was responsible for the infarcts of the spleen. My inclination is to believe it was the endocarditis. One other finding of significance was an organizing pulmonary infarct 5 cm. in diameter in the right upper lobe.

*Doan, C. A., and Wiseman, B. K.: The monocyte, monocytosis, and monocytic leukosis: a clinical and pathological study. Ann. Int. Med. 8:383, 1934.

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SULFANILAMIDE

The need for caution by physicians in the use of prontosil, prontylin and other drugs of the sulfanilamide group has been commented on repeatedly in the columns of this *Journal* (215:1311, 1936; 216:706 and 1138, 1937). The reports of the brilliant therapeutic results following the use of these compounds are outstanding articles in medical literature for the current year. Infections due to hemolytic streptococci, meningococci, gonococci and even the gas bacillus appear to be equally susceptible — so much so that the enthusiastic and optimistic practitioner is apt to neglect well-recognized and essential procedures other than treatment with the drug and to forget that sulfanilamide may be extremely toxic in a given case.

Little in the way of specific recommendations can be made concerning general methods of therapeutic procedure, for they are determined by the individual case. One can only say that sensible and conservative principles, both medical and surgical, should be combined with the use of the drug.

More detailed information can be given in regard to toxic manifestations. In the first report in the English literature the occurrence of sulfhemoglobinemia was noted in several cases. Since then granulocytopenia — a reduction of the circulating, white blood cells derived from the bone marrow — and anemia of such severe grades as to warrant discontinuing the drug have been reported. In addition a case of toxic optic neuritis and several of annoying skin eruptions have been observed. All such untoward results clearly indicate that some individuals are particularly susceptible to the drug and that every patient to whom it is given must be carefully watched for signs of intoxication.

An editorial in a recent issue of the *Journal of the American Medical Association* (109:1128, 1937) warns against the potential hazards of sulfanilamide. Magnesium sulfate and some of the coal-tar derivatives should not be administered concurrently. (Such incompatibility may be the explanation of the recent deaths — over fifty — following the use of an "elixir of sulfanilamide" — a solution of sulfanilamide in diethylene glycol.) The same editorial also cautions the practitioner to exercise extreme care in prescribing the various proprietary derivatives that will inevitably appear on the market. Even though the formula of such a derivative closely approximates that of sulfanilamide, the new drug may be many times more toxic.

Once again, we caution against the indiscriminate use of a drug which gives every indication of being one of the most important ones ever made available to the medical profession.

ETIOLOGY OF CANCER

An addition to the many theories of the etiology of cancer has recently been made by Albert Fischer.[1] His long experience in the field of tissue growth entitles his views to most serious consideration. On the basis of repeated transplants of mammary tissue in mice which led to the development of carcinoma of varying degrees of malignancy, he believes that there are scattered malignant cells with unlimited ability to grow and multiply within almost all portions of the organism. The malignant tumor cells, then, are but a rare variety of the normal tissue cell and as such are present in the tissues and organs of every individual. Probably very few in number, they do not ordinarily proliferate to form tumors. But by autologous grafting of normal tissue it is possible to render more favorable the conditions for development of these scattered, potentially neoplastic cells. In one hundred and fifty transplants of mammary tissue fifteen tumors were obtained.

There are several points of interest in this theory which demand most careful investigation. Autologous and homologous transplants have been made for years in animals by Leo Loeb without noting the development of tumor. Furthermore, autologous transplants have been repeatedly made in man through such diverse procedures as gastroenterostomy, skin grafting and the spontaneous process of endometriosis. In all these instances neoplasms have not been encountered. Since market mice were used in these experiments by Fischer, it is quite possible that the stock was such that the mammary tissue transplanted actually possessed neoplastic tendency. Spontaneous carcinoma of the breast is very frequent in many strains of mice, and an incidence of ten per cent would not be at all surprising. Moreover, although Fischer included portions of the tail of the mouse in the transplants in these experiments, in no instance were there tumors of stratified squamous epithelium, of bone or of connective tissue. In other words, the only types of tumor developing from these transplants were those which might well have appeared in the breast had it been left in its normal site.

It is as yet too soon to evaluate these most interesting experiments and the hypothesis to which they have led. Until they are thoroughly established and until the various possible difficulties involved are evaluated to the full, conclusions should be avoided.

REFERENCE

1. Fischer, A.: The theory of the developmental physiology of malignant tumors. Am. J. Cancer 31:1, 1937

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

M. Fletcher Eades, M.D., *Secretary*
19 Bay State Road
Boston

Case History No. 44. Bleeding at Six Months

Mrs. C. L., aged thirty-two years, entered the hospital July 5, 1934, having definite uterine contractions and slight staining. She was six months pregnant by dates, and definite fetal movements could be made out. The pains continued throughout the day, and during the afternoon staining became more profuse.

There was no family history of tuberculosis, diabetes, malignancy or hemorrhagic diseases. The patient's health had always been good. She had had one previous full-term pregnancy with a normal delivery and no complications. She had no history of hypertension or kidney disease. The last menstrual period was in January, 1934.

On physical examination the patient appeared well nourished. Her weight was 172 pounds. Her blood pressure was 120 systolic, 70 diastolic. There was no nausea or vomiting. Her temperature was 98.6°F., and the pulse was 80. She was in excellent condition and showed no sign of having lost much blood. The urine was essentially negative.

The antepartum course was uneventful until the day of entry to the hospital, when the patient complained of definite uterine contractions and slight staining. On vaginal examination the membranes were found to be bulging into the vagina. The cervix was dilated so as to admit three or four fingers, and was partly taken up. The edge of the placenta was on the posterior border of the cervix and covered about one third of the internal os. The membranes were ruptured; this brought the pressure of the head on to the placenta and stopped the bleeding.

For the next twenty-four hours the patient had

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

irregular labor with very little progress. The pains were fairly good at times and weak at others. There was slight staining during this time.

The patient was again examined vaginally on the next day, and the cervix was found to be about three-quarters dilated and easily admitted the hand. The vertex was presenting. One foot of the baby was pulled down, and the head pushed up. A slow breech extraction was done with moderate ease. Considerable difficulty was encountered in extracting the head by pressure from above, due to incomplete dilatation of cervix. The baby cried immediately on birth. It weighed one pound and fourteen ounces. There was a very slight tear of the posterior cervix, which did not require sutures. The placenta and membranes were delivered intact. The patient was in good condition. The baby lived for about twelve hours in the incubator, but gradually became blue and died from prematurity.

The mother had an uneventful postpartum course and returned home after eleven days in the hospital.

Comment. This case is of interest in showing a placenta previa causing hemorrhage and labor before the period of viability. The majority of cases do not manifest themselves until after the seventh month. Unquestionably there are cases throughout the second trimester in which a so-called miscarriage or premature labor is due to this cause. The symptoms, especially the hemorrhage, are apt to be of such minor significance that the diagnosis is not considered. The earlier in pregnancy a placenta previa asserts itself the less the symptoms and danger.

In regard to the treatment of this case, delivery from below was the only reasonable answer. The cervix was partly dilated and the membranes were bulging into the vagina when the patient was seen. There was no hope of saving the pregnancy at this point. Rupture of the membranes was indicated to hasten expulsion of the fetus. When the diagnosis of placenta previa was made, rupture of the membranes was indicated to control the bleeding. If the bleeding was not controlled by this procedure a bag could have been inserted to put pressure on the placenta. Another and often preferable method is a Braxton Hicks version with sufficient traction on the fetus to control bleeding. However, in this case the cervix was sufficiently dilated to deliver the body of the fetus. The difficulty of delivery came with the passing of the after-coming head. This is the dangerous point in such cases and is often the cause of severe and dangerous lacerations of the cervix. If the cervix is not dilated, the patient should be treated conservatively by rest in bed and sedatives. In the absence of profuse hemorrhage there is no indication for immediate treatment. If the bleeding stops and the patient seems able to carry on with her pregnancy, an examination for placenta previa should be made before the patient is allowed to leave the hospital.

MISCELLANY

MAINE NEWS

Clinical Session, Maine Medical Association

A clinical session of the Maine Medical Association was held in Portland on October 21 and 22. Surgical and medical demonstrations were given at the Maine General Hospital, Maine Eye and Ear Infirmary, Children's Hospital and St. Barnabas Hospital. A reception for all members at the home of Dr. E. G. Abbott on the afternoon of October 21 was followed by dinner at the Eastland Hotel, at which the principal address was given by Dr. Milton C. Winternitz, professor of pathology, Yale University School of Medicine, on the subject "The Pathology of Vascular Diseases."

A luncheon meeting of the Council was held on October 21 in the directors' office of the Maine General Hospital. The county secretaries met at luncheon on October 22 at the Maine Eye and Ear Infirmary.

BROOKFIELD MEDICAL CLUB

The fifty-fifth annual meeting of the Brookfield Medical Club was held at the Brookfield Inn, Wednesday, October 20, with the retiring president, Dr. John E. Dalton, of Warren, host, and Dr. John M. Birnie, of Springfield, guest speaker. The latter's subject was "Salt, and Its Influence in Metabolism."

The following officers were elected for the coming year: Dr. Alphonse H. Petit, of Ware, president; Dr. James M. Murphy, of Palmer, vice-president; Dr. John R. Fowler, of Spencer, secretary and treasurer; Drs. William F. Hayward, Thomas Keeley, and Joseph Slowick, executive committee.

J. R. Fowler, M.D., *Secretary.*

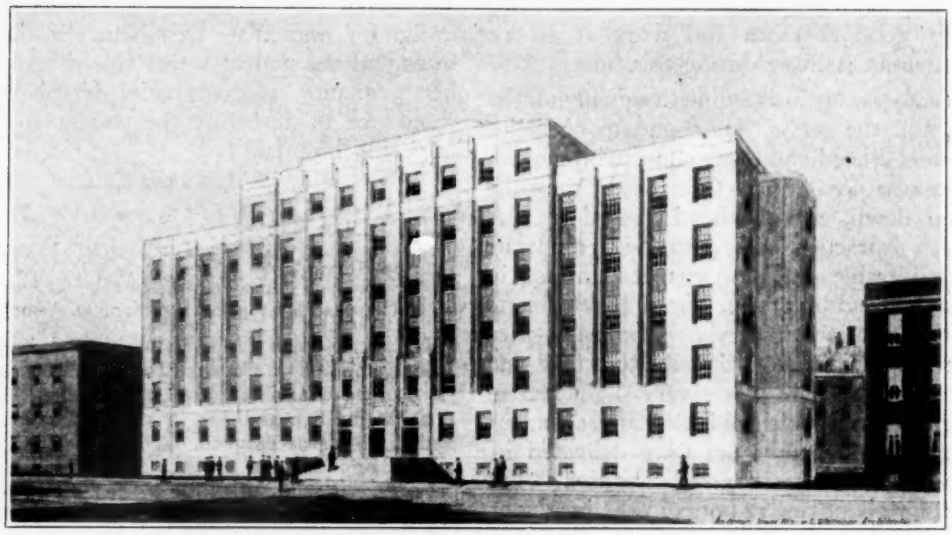
GOLFERS' SPECIAL

A "Golfers' Special" to the San Francisco meeting of the American Medical Association is being organized by the American Medical Golfing Association. Physicians who like golf mixed with their travel will find five games arranged on the trip out to the coast and three games on the return trip through the Northwest. The first game will be played in New Orleans, reached by the *S.S. Dixie,* from New York (or via a rail itinerary), on Tuesday, June 7. Other stops include Houston, Galveston and San Antonio, Texas; Los Angeles and Del Monte, California; and finally San Francisco where the big A. M. G. A. tourney will be held on Monday, June 13.

The return trip includes Portland, Oregon; Seattle, Washington; Vancouver, B. C.; Lake Louise and Banff; and finally St. Paul and Chicago.

Nongolfers as well as golfers, and their ladies, are welcome and will find the A. M. G. A. Special a glorious experience.

For full particulars write Dr. Walt P. Conway, 1723 Pacific Avenue, Atlantic City, N. J., the president of the A. M. G. A.; or Bill Burns, executive secretary, 2020 Olds Tower, Lansing, Michigan.



NEW TUFTS COLLEGE MEDICAL SCHOOL

Plans were recently released for the new Tufts College Medical School building to be erected at the New England Medical Center, Bennet Street, Boston, with funds to be obtained from the $2,000,000 campaign that has just been launched.

The building, designed in modern style by Andrews, Jones, Biscoe and Whitmore, Boston architects, will be a six-story structure of brick and stone, with a frontage of about 225 feet. All the latest developments in medical school construction have been incorporated in the plans.

Ample space is provided for all departments. The first floor will be given over to administrative offices, library and students' lounge. The physiology and pharmacology departments will occupy the second floor, with pathology and bacteriology on the third, biochemistry and clinical medicine on the fourth, anatomy and histology on the fifth, and the anatomy museum on the sixth floor. Each floor will have its lecture room, research laboratories and departmental library.

It is estimated that the new building will cost in the neighborhood of $750,000.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR AUGUST, 1937

DISEASES	August 1937	August 1936	Five-Year Average*
Anterior Poliomyelitis	147	5	132
Chickenpox	105	118	110
Diphtheria	17	19	55
Dog Bite	780	1063	885
German Measles	26	45	40
Gonorrhea	490	562	621
Lobar Pneumonia	120	126	102
Measles	107	230	190
Meningococcus Meningitis	11	6	4
Mumps	95	207	168
Scarlet Fever	112	156	219
Syphilis	528	462	395
Tuberculosis, Pulmonary	303	281	317
Tuberculosis, Other Forms	46	29	35
Typhoid Fever	12	13	21
Undulant Fever	3	11	3
Whooping Cough	682	533	512

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from Adams, 3; Amesbury, 4; Auburn, 1; Barnstable, 1; Beverly, 8; Boston, 24; Brockton, 1; Brookline, 1; Cambridge, 1; Chelsea, 1; Danvers, 3; Dartmouth, 1; Dedham, 1; Framingham, 1; Gloucester, 3; Hamilton, 2; Haverhill, 4; Ipswich, 5; Lexington, 1; Lynn, 7; Malden, 2; Manchester, 1; Marblehead, 2; Maynard, 1; Medford, 1; Melrose, 1; Merrimac, 2; Milford, 1; Millbury, 1; Milton, 2; Montague, 1; Natick, 1; New Bedford, 2; Newburyport, 7; Newton, 4; North Adams, 1; Norwood, 1; Pittsfield, 6; Plainfield, 1; Quincy, 6; Randolph, 1; Reading, 2; Revere, 1; Rowley, 1; Salem, 3; Salisbury, 1; Saugus, 1; Shelburne, 1; Somerville, 3; Springfield, 1; Stockbridge, 1; Swampscott, 1; Topsfield, 1; Wakefield, 1; Waltham, 2; Watertown, 1; West Newbury, 1; West Springfield, 1; Williamstown, 2; Winchester, 1; Worcester, 3; total, 147.

Diphtheria was reported from Boston, 1; Lexington, 2; New Bedford, 2; Quincy, 1; Salem, 1; Tewksbury State Infirmary, 10; total, 17.

Dysentery, bacillary was reported from Beverly, 3; Boston, 3; Waltham, 14; total, 20.

Encephalitis lethargica was reported from Danvers, 1; Revere, 1; total, 2.

Malaria was reported from Boston, 1; Foxboro, 2; Gloucester, 1; Lancaster, 1; Medford, 1; total, 6.

Meningococcus meningitis was reported from Boston, 1; Clinton, 1; Dudley, 1; Fitchburg, 1; Longmeadow, 1; New Bedford, 1; Newton, 1; Northampton, 1; Norwood, 1; Quincy, 1; Springfield, 1; total, 11.

Paratyphoid "B" was reported from Amesbury, 1; Boston, 8; Bridgewater, 1; Brookline, 1; Easthampton, 1; Easton, 1; Lowell, 4; Lynn, 2; Malden, 2; Manchester, 1; Melrose, 1; Newton, 1; Northampton, 3; Sandwich, 1; Stoneham, 1; Waltham, 1; Westfield, 1; West Springfield, 1; Winchester, 1; Winthrop, 1; total, 34.

Septic sore throat was reported from Boston, 2; Lawrence, 1; Mansfield, 1; Marlboro, 1; Newburyport, 1; Pittsfield, 1; total, 7.

Trachoma was reported from Dudley, 1; Pittsfield, 1; West Springfield, 1; total, 3.

Trichinosis was reported from Boston, 2; Maynard, 1; total, 3.

Typhoid fever was reported from Boston, 3; Cambridge, 1; Holyoke, 1; Lowell, 1; New Bedford, 1; Quincy, 1; Somerville, 2; Springfield, 1; Worcester, 1; total, 12.

Undulant fever was reported from Chicopee, 1; Nantucket, 1; North Adams, 1; total, 3.

Diphtheria continued to show a record low incidence for the eleventh consecutive month.

The incidence of meningococcus meningitis was again above the five-year average.

The reported incidences of measles, mumps, German measles, pulmonary tuberculosis and scarlet fever were below the five-year average.

The reported incidence of typhoid fever continued to show a record low figure for any year to date.

Paratyphoid fever for the fifth consecutive month had the highest reported incidence both for August and any year to date.

The incidence of chickenpox fell from a record high in July to a figure below the five-year average.

The reported incidence of whooping cough continued above the five-year average.

Lobar pneumonia and tuberculosis (other forms) were reported above the five-year average.

The incidence of undulant fever was not remarkable.

The reported incidence of anterior poliomyelitis was slightly above the five-year average.

Although the incidence of animal rabies decreased in comparison with that of July, the distribution of cases was still unusually wide throughout the state. Previous foci in the vicinity of Billerica, Woburn and the North Shore were active. New foci appeared in Winchester, Rowley, Holbrook and Danvers.

CONNECTICUT NEWS

Litchfield County Medical Association

The Litchfield County Medical Association met for its fall meeting at the camp of its councilor, Dr. Harry B. Hanchett, on Bantam Lake, Tuesday, October 5. As has been the custom for years, this semiannual gathering of the oldest medical society in the country was characterized by an informal steak dinner. The pièce-de-résistance was cooked over an open outdoor fire by Mrs. Hanchett and Dr. Thomas L. Thomson. There was no lack of good will and friendliness, but the usual mirth and wit were noticeably lacking due to the absence of Dr. Charles Turkington, president of the Connecticut State Medical Society and famed general practitioner of Litchfield. We mourn with Dr. Charlie the loss of his father, octogenarian and sheriff of Litchfield County.

Dr. Fuldner from the State Department of Health presented an outline of the work for crippled children in Connecticut under the Social Security Act. The chairman of the Council of the State Society, Dr. James Douglas Gold, brought a message of activity from the Council. The editor-in-chief of the *Journal*, Dr. Stanley B. Weld, touched a few high spots in medical news of today and appealed for a more active use of the *Journal*. Past president of the Society, Dr. Daniel C. Patterson, of Bridgeport, related some interesting surgical cases from his personal experience.

Dr. Harry's steak dinners on the shore of Bantam Lake among the brilliant autumn foliage are an institution in themselves peculiar to this corner of Connecticut.

Fairfield County Medical Association

The semiannual meeting of the Fairfield County Medical Association was held at the Woodway Country Club, Springdale, on October 6. A large number of the members and guests enjoyed an afternoon of golf. At the dinner meeting in the evening, delegates from other county associations and guests were introduced.

"The Pathology of Arteriosclerosis" was presented by Dr. Milton C. Winternitz, professor of pathology at Yale University School of Medicine. Dr. Winternitz's paper covered an extremely interesting piece of research in the field of pathology. An attendance of 130 broke all records for the association in recent years.

Hartford Hospital Receives $2,000 Gift

The will of the late Dr. Edward K. Root, of Hartford, which has been filed for probate, contains gifts of his professional library and instruments to the Hartford Medical Society and a bequest of $2,000 to the Hartford Hospital to be known as "The Edward K. Root Fund." The income from this fund is to be used for providing medical periodicals for the house staff of the hospital.

Three Hospitals Recipients of Gifts

Commemorating his eighty-fifth birthday on September 28, George A. Gay, one of Hartford's best-known retired merchants, announced gifts totaling $20,000 to hospitals and philanthropic organizations in the vicinity of Hartford. Among these gifts were the following: Hartford Hospital, $10,000; St. Francis Hospital, $5,000; Newington Home for Crippled Children, $2,000.

Hospital Building Program for Hartford

A $150,000 building program to meet urgent institutional needs at the Municipal Hospital, Hartford, was decided on recently by the Public Welfare Commission. The majority of this amount will be used in building a new brick wing for the Isolation Hospital and remodeling one of the old wooden buildings for use as an operating room and surgery.

Deaths

LOVELAND — John Elijah Loveland, M.D., seventy-two, of Middletown, died suddenly on September 12 at his summer home in Old Saybrook. Dr. Loveland at the time of his death was medical examiner in Middletown and had been prominent in the advancement of Middlesex Hospital in that city.

THOMPSON — George Thompson, M.D., seventy-six, a retired physician and former resident of Norwich, died September 17 after a long illness. He was born in Union, Maine, graduated from Bowdoin College and Bowdoin Medical School and took up practice in Norwich, where he was a member of the staff of William W. Backus Hospital. He practiced in Taftville and Norwich for forty-four years. In 1920 he was elected to the legislature from Norwich.

He is survived by his wife, Mrs. Mary Greene Thompson, a daughter, Miss Helen Thompson, of Norwich, and a son, Dr. Hartwell G. Thompson, of Hartford, with whom he lived.

CORRESPONDENCE

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of September 7 the following have been accepted:

Chappel Bros., Inc.
Vials Chappel Liver Extract (Subcutaneous) 10 cc.

Eli Lilly & Co.
Pulvules Pentobarbital-Sodium — Lilly, ¾ grain
Suppositories Pentobarbital-Sodium — Lilly, 2 grains

Mallinckrodt Chemical Works
Mandelic Acid

Monsanto Chemical Co.
Sulfanilamide

National Drug Company
Ampul-Vials Solution Sodium Morrhuate 5% with Benzyl Alcohol 2%, 5 cc. size
Ampul-Vials Solution Sodium Morrhuate 5% with Benzyl Alcohol 2%, 25 cc. size
Ampul-Vials Solution Sodium Morrhuate 10% with Benzyl Alcohol 2%, 25 cc. size
Mixed Grass Pollen Antigen — National
Sulfanilamide
Tablets Sulfanilamide, 5 grains

Sharp & Dohme
Propadrine Hydrochloride
Propadrine Hydrochloride Capsules, ⅜ grain (0.024 gm.)
Propadrine Hydrochloride Nasal Jelly, 0.66%

E. R. Squibb & Sons
Grasses Combined Pollen Allergen Solutions — Squibb, 5 cc. vials
Ragweed Combined Pollen Allergen Solutions — Squibb, 5 cc. vials

The following article has been accepted for inclusion in the list of articles accepted by the Council but not described in N.N.R. (New and Nonofficial Remedies, 1937, p. 483):

The Dermo Co.
Casil Protective Skin Creme

PAUL NICHOLAS LEECH, *Secretary.*
535 North Dearborn Street,
Chicago, Illinois.

RECENT DEATH

KEENAN — GEORGE F. KEENAN, M.D., of 207 Bay State Road, Boston, died October 26, in St. Elizabeth's Hospital. He was in his fifty-ninth year.

Born in Cambridge, he received his education at the Phillips Grammar School, English High School, Massachusetts Agricultural College and the Tufts College Medical School. Shortly after his graduation from medical school, Dr. Keenan served at the Haymarket relief station and the Boston City Hospital. In 1908 he was made resident surgeon of the East Boston relief station and was chief surgeon to St. Elizabeth's Hospital at the time of his death.

During his twenty years of military service in the national guard and in the regular army, beginning in the Spanish-American War, he rose from the rank of private to that of brigadier-general. During the World War, he served as surgeon of the "Dixie" division and as chief medical officer of the second army corps. Dr. Keenan retired from the army in 1921.

His memberships included the Tufts College Medical Alumni Association, of which he was at one time president. He held fellowships in the Massachusetts Medical Society, the American Medical Association and the American College of Surgeons.

His widow, and a son survive him.

NOTICES

REMOVAL

LESLIE H. VAN RAALTE, M.D., announces the removal of his office to 1230 Hancock Street, Quincy.

ANNOUNCEMENT

EUGENE FRANCIS CALLANAN, M.D., announces the opening of an office at 482 Commonwealth Avenue, Boston. Telephone: Kenmore 5170.

GREATER BOSTON MEDICAL SOCIETY

The list of speakers on the 1937-1938 program of the Greater Boston Medical Society is as follows:

December 7 — George Baehr, M.D., Mt. Sinai Hospital, New York.
January 4 — Symposium of Oto-Laryngology, Beth Israel Hospital.
February 1 — Max Cutler, M.D., Michael Reese Hospital, Chicago.
March 1 — Edward D. Churchill, M.D., Massachusetts General Hospital.
April 5 — Louis Nahum, M.D., New Haven, Connecticut.
May 3 — Annual meeting and dinner.

All meetings except that of May will be held in the auditorium of the Beth Israel Hospital.

KERMIT C. ROSEN, M.D., *President,*
DAVID B. STEARNS, M.D., *Secretary.*

BOSTON CITY HOSPITAL

CLINICOPATHOLOGICAL CONFERENCE

The monthly clinicopathological conference will be held at the hospital on Wednesday, November 10, at 12 o'clock noon, in the Pathological Amphitheater.

JOSEPH E. HALLISEY, M.D.,
Secretary, Medical Staff.

RADIO BROADCASTS

The second group of weekly broadcasts sponsored by the American Medical Association and the National Broadcasting Company concern hygiene. These dramatized health messages are intended to furnish supplementary material for health teaching in junior and senior high schools and are broadcast every Wednesday from 2:00 to 2:30 p. m. over the Red Network. The dates and subjects are as follows:

November 10 — Playing for Fun. Health values and hazards in sports and recreation, including football.
November 17 — Fresh Air, Fresh Clothes and Fresh Skin. Ventilation; clothing; bathing.
November 24 — Rest, Relaxation, Refreshment. All

work and no play, or all play and no work — bad for health.

December 1 — Tuberculosis, Foe of Youth. How bad habits of hygiene and unwise living, plus infection, favor tuberculosis.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, November 16, at 12 noon. The speaker will be Alton S. Pope, M.D., director, Division of Tuberculosis, Massachusetts Department of Public Health. His subject will be "Prevention of Tuberculosis." All physicians are cordially invited to attend.

AMERICAN ACADEMY OF PEDIATRICS

PROGRAM OF THE MEETING OF REGION 1 MEMBERS, FRIDAY AND SATURDAY, NOVEMBER 12 AND 13

Morning Session, Friday, November 12, 9:30 a. m. in Amphitheater E of the Harvard Medical School

SYMPOSIUM: CERTAIN RELATIONS OF VITAMIN C TO NUTRITION

The Recognition of Early Deficiency States. Dr. Kenneth D. Blackfan.

The Fundamental Pathology of Vitamin C Deficiency. Dr. S. B. Wolbach.

The Use and Abuse of Vitamin C Determinations. Dr. Otto A. Bessey.

The Relation of Vitamin C Deficiency to Dental Lesions. Dr. Paul E. Boyle.

Ascorbic Acid Values in Pregnancy. Dr. Harold M. Teel.

Ascorbic Acid Metabolism in Early Infancy. Dr. Theodore H. Ingalls.

The Relation of Vitamin C Deficiency to Wound Healing. Dr. Thomas H. Lanman.

CLINICAL DEMONSTRATION

Effects of Anemia Upon Growth. Dr. Louis K. Diamond.

* * *

Luncheon: 1 p. m., at Vanderbilt Hall, the Harvard Medical School Dormitory

* * *

Afternoon Session, Friday, November 12, 2:30 p. m. in Amphitheater E of the Harvard Medical School

Acidosis, Alkalosis and Dehydration. Dr. James L. Gamble.

Pyelonephritis and Hypertension. Dr. Allan M. Butler.

The Late Stage of Pyelitis and Pyelonephritis as Seen in Adult Life. Dr. Soma Weiss and Dr. Frederic Parker, Jr.

Estrogenic Factors in Early Adolescence. Dr. Ira Nathanson and Dr. Joseph C. Aub.

Syndrome Characterized by Osteitis Fibrosa, Pigmentation and Gonadal Dysfunction. Dr. Fuller Albright.

Some Laboratory Studies in Poliomyelitis. Dr. James D. Trask.

The Normal Development of Infants' Feet. Dr. William T. Greene.

Congenital Malformations of the Intestinal Tract. Dr. William E. Ladd.

* * *

Dinner: 6:30 p. m., at the Copley Plaza Hotel

Morning Session, Saturday, November 13, 9 a. m. in Amphitheater E of the Harvard Medical School

SYMPOSIUM: FUNCTIONAL ANOMALIES IN VISION OF CHILDREN

Growth and Development of the Visuo-Sensory Mechanism. Dr. Theodore L. Terry.

Amblyopia in Children. Dr. Paul A. Chandler.

Growth and Development of the Visuo-Motor Mechanism. Dr. Virgil G. Casten.

Strabismus. Dr. Walter B. Lancaster.

Refractive Errors in Children. Dr. Harry K. Messenger.

What Constitutes an Adequate Eye Examination in Children? Dr. J. Herbert Waite.

Factors Influencing the Incidence and Course of Otitis Media in Scarlet Fever. Dr. Conrad Wesselhoeft.

* * *

Luncheon: 1 p. m., at Vanderbilt Hall, the Harvard Medical School Dormitory

* * *

Saturday Afternoon Session, November 13

One hour clinical demonstrations will be given at 2:30 p. m. and repeated at 4 p. m. Attendance is limited.

1. Symposium: Rheumatic Heart Disease.
   Observations on the Etiology of Rheumatic Fever — Dr. T. Duckett Jones.
   Present Status of 1,000 Rheumatic Fever and Chorea Patients Followed an Average of Ten Years — Dr. Edward F. Bland.
   Observations on the Natural History of Rheumatic Fever — Dr. John P. Hubbard.
   Experiences with Sulfanilamide — Dr. Benedict F. Massell — House of the Good Samaritan, 25 Binney Street, Boston.
2. Lung Pathology in Children — Dr. Harold L. Higgins — Massachusetts General Hospital, Fruit Street, Boston.
3. Blood Studies in Rheumatic Fever — Dr. Elmer W. Barron and Staff — Boston Floating Hospital, 20 Ash Street, Boston.
4. Studies in Child Development. Demonstrations by pediatrician, orthopedist and others — Dr. Harold C. Stuart and Staff — Clinic of the School of Public Health, 316 Longwood Avenue, Boston.
5. The Early Treatment of Anterior Poliomyelitis. Cases — Dr. Arthur T. Legg — Children's Hospital, 300 Longwood Avenue, Boston.
6. Hemorrhagic Disturbances in Childhood — Dr. Charles F. McKhann — Children's Hospital.
7. Lantern Slide Demonstration of Infant and Child Pathology — Dr. Sidney Farber — Children's Hospital.
8. Studies of Children After Infections Involving the Central Nervous System. Cases — Dr. Elizabeth Lord and Dr. Bronson Crothers — Children's Hospital.

Instead of the above, one of the following visits may be made at 2:30 p. m.

9. Wrentham State School, Wrentham, Mass. — Dr. C. Stanley Raymond. This is an unusual opportunity to see a large group of mentally defective children uniquely classified and demonstrated.

10. Massachusetts Institute of Technology, Memorial Drive, Cambridge. The Institute invites members to visit its laboratories where some recent scientific developments of pediatric interest will be shown.

11. The Gardner Museum on the Fenway, and the Boston Museum of Fine Arts, 465 Huntington Avenue, Boston. These museums are both near the Children's Hospital.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, November 8

Monday, November 8

5 p. m. Edward K. Dunham Lecture. Harvard Medical School, Amphitheater, Building C.

Tuesday, November 9

•9 a. m. - 10 a. m. Boston Dispensary. Contrasts in the Management of Tuberculosis and Syphilis. Dr. William A. Hinton.

Wednesday, November 10

•9 a. m. - 10 a. m. Boston Dispensary. Hospital Case Presentation. Dr. S. J. Thannhauser.

•12 m. Clinicopathological conference. Children's Hospital Amphitheater.

12 m. Boston City Hospital. Clinicopathological Conference. Pathological Amphitheater.

5 p. m. Edward K. Dunham Lecture. Harvard Medical School, Amphitheater, Building C.

Friday, November 12

9:30 a. m. American Academy of Pediatrics. All-day meeting.

•9 a. m. - 10 a. m. Boston Dispensary. Lung Abscess. Dr. Frederick T. Lord.

5 p. m. Edward K. Dunham Lecture. Harvard Medical School, Amphitheater, Building C.

Saturday, November 13

9 a. m. American Academy of Pediatrics. All-day meeting.

•9 a. m. - 10 a. m. Boston Dispensary. Hospital Case Presentation. Dr. S. J. Thannhauser.

•10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

•Open to the medical profession.

November 4 — Faulkner Hospital, Clinicopathological Conference, 5 p. m.

November 6 — American Board of Obstetrics and Gynecology. Page 153, issue of July 22, and page 609. February 5 — Page 609. June 13 and 14 — Page 153, issue of July 22 and page 609, issue of October 7.

November 7 - March 6 — Public lectures at Beth Israel Hospital. Page 685, issue of October 21.

November 8, 10, 12 — The Edward K. Dunham lectures. Page 721, issue of October 28.

November 10 — Boston City Hospital, Clinicopathological Conference. Page 762.

November 12 and 13 — American Academy of Pediatrics. Page 763.

November 16 — South End Medical Club. Page 763.

November 18 — Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, 8:30 p. m.

December 7 - May 3 — Greater Boston Medical Society. Page 762.

April 4-8, 1938 — The American College of Physicians. Page 41, issue of July 1.

District Medical Societies

BRISTOL SOUTH

November 4 — 5 p. m., Fall River.

May 5, 1938 — 5 p. m., New Bedford.

ESSEX NORTH

November 4 — Censors' meeting, 4 p. m. Hotel Bartlett, Haverhill.

ESSEX SOUTH

November 4 — Censors meet at Salem Hospital, 3:30 p. m.

December 1 — Salem Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Leland S. McKittrick. Subject: Carcinoma of Colon. Discussion of Diagnosis.

January 5 — Danvers State Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

February 2 — Council Meeting, Boston.

February 9 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John B. Hawes, 2nd. Subject: Dust and Disease.

March 2 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

April 6 — Gloucester Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

May 5 — Censors meet at Salem Hospital, 3:30 p. m.

May 11 — Annual meeting, Salem Country Club, Peabody. Dinner at 7 p. m. Speaker and subject to be announced.

FRANKLIN

Meetings will be held at the Franklin County Hospital, Greenfield, at 11 a. m. the second Tuesdays of November, January, March and May.

HAMPDEN

November 4 — Censors' meeting. Page 685, issue of October 21.

Other meetings will be held on the fourth Tuesday in January, April and July.

MIDDLESEX EAST

Meetings will be held at the Bear Hill Golf Club, Stoneham, at 12:15 p. m. on November 17, January 12, March 16, and May 11.

MIDDLESEX NORTH

Meetings will be held at the Vesper Country Club, Lowell, on January 26, and April 27.

NORFOLK DISTRICT

November 30 — Hotel Kenmore. 8:15 p. m. Diagnosis of Lesions of the Hand with Demonstration of Patients. Dr. William E. Browne.

January 25 — Hotel Kenmore. 8:15 p. m. The Management of Uterine Prolapse. Dr. Louis E. Phaneuf.

February 23 — Hotel Kenmore. 8:15 p. m. Dermatitis Venenata Due to Cosmetics and Industrial Irritants. Dr. John G. Downing. Discussion by Dr. Francis P. McCarthy.

March 29 — Hotel Kenmore. 8:15 p. m. Subject to be announced but to be related to diseases of the kidney. Dr. Albert A. Hornor.

May, 1938 — Annual meeting.

The censors meet on the first Thursdays of May and November in each year.

NORFOLK SOUTH.

November 4 — Page 722, issue of October 28.

PLYMOUTH

Meetings will be held at 11 a. m. on November 18, January 20, March 17, April 21, May 19 and July 21.

SUFFOLK

November 4 — Censors' meeting. Page 685, issue of October 21.

November 17 — Joint meeting with Boston Orthopedic Society.

January 19 — Joint meeting with Boston Medical Library.

March 15 — Joint meeting with Boston Obstetrical Society.

WORCESTER

At the following meetings, except the annual meeting, dinner will be at 6:15, to be followed by business session and scientific program.

November 10 — Grafton State Hospital, North Grafton.

December 8 — St. Vincent Hospital, Worcester.

January 12 — Worcester City Hospital, Worcester.

February 9 — Worcester State Hospital, Worcester.

March 9 — Memorial Hospital, Worcester.

April 13 — Hahnemann Hospital, Worcester.

May 11 — Afternoon and evening, annual meeting. Place and schedule of program to be announced.

BOOKS RECEIVED FOR REVIEW

Biological and Clinical Chemistry. Matthew Steel. 770 pp. Philadelphia: Lea & Febiger, 1937. $8.00.

Radiation Therapy: Its use in the treatment of benign and malignant conditions. Oxford Medical Publications. Ira I. Kaplan. 558 pp. New York: Oxford University Press, 1937. $10.00.

The Management of the Pneumonias: For physicians and medical students. Oxford Medical Publications. Jesse G. M. Bullowa. 508 pp. New York: Oxford University Press, 1937. $8.50.

The Human Mind. Karl A. Menninger. Second edition. 504 pp. New York: Alfred A. Knopf, 1937. $5.00.

Dr. Colwell's Daily Log for Physicians: A brief, simple, accurate financial record for the physician's desk. Champaign, Ill.: Colwell Publishing Company, 1937. $6.00.